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THE GENETICS OF THE DOG

THE GENETICS OF THE DOG

by

MARCA BURNS, B.Sc.

Technical Communication No. 9
of the Commonwealth Bureau of
Animal Breeding and Genetics
Edinburgh

COMMONWEALTH AGRICULTURAL BUREAUX
FARNHAM ROYAL
SLOUGH BUCKS

FIRST PUBLISHED - 1952

PRINTED IN GREAT BRITAIN
BY ROBERT CUNNINGHAM AND SONS LTD., LONGBANK WORKS, ALVA

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ACKNOWLEDGMENTS

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Finally, I cannot fail to acknowledge the constant inspiration and instruction provided by my many canine friends of all breeds and mixtures, and especially by those irrepressible collaborators, the 'Questings'.

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I

INTRODUCTION—MENDELIAN THEORY

A VAST amount of information on the subject of canine inheritance is scattered through the innumerable dog books and canine periodicals, but it is not necessary to attempt the impossible task of summarising this popular literature, since it is readily available to every breeder; the interested fancier will obtain for himself the books relevant to his breed or breeds, and will do his best, in the light of his own experience, to winnow the wheat from the chaff. With scientific literature the position is different: because of the technical language in which it is written, and the specialised journals in which it is published, it is very inaccessible to non-scientists. The object of this book is therefore to present scientific knowledge of inheritance in the dog in a form accessible to practical dog breeders, and the information offered here is nearly all derived from scientific papers or books published by research workers. It would not be possible to review the work of scientists without using technical terms to some extent, but it is hoped that those used are adequately explained, and that the book will not be too difficult to follow if the reader will make the effort which is inevitably necessary for the comprehension of any complicated subject. Throughout the book, the interests of the British and Commonwealth dog breeding fraternity have been particularly considered, but research work from all over the world is reviewed, and it is hoped that the book will be found useful by dog breeders and scientists outside the Commonwealth as well as within it. There is, of course, a great deal more to scientific dog breeding than can be included in a book on genetics. Science has much to say about other important subjects, such as feeding, hygiene, training, and, of course, veterinary matters, for information on which other specialist books must be consulted.

As dog breeders, what we require to know is how breeds, and strains within breeds, change or can be changed, and how, despite the fact that no two individuals are ever exactly alike, we can nevertheless obtain a large proportion of animals which resemble each other in having the good qualities which we desire. The attempt to attain such knowledge may take the form of studies on general breeding methods, such as the effects of inbreeding and outcrossing, the influence of pedigree, feeding or training on general type or behaviour, etc.; or it may take the form of observing the inheritance of particular clearly-defined attributes such as coat-colour, dew-claws, or ear-carriage, in the descendants of animals which differed sharply from each other in the attribute under consideration. Scientists have contributed knowledge of both these kinds, but by far the majority of their studies on dogs have been of the second kind, and it is in this type of work that the Laws of Mendel are most useful. It should be remembered that the Laws of Mendel only work out in practice where a few clearly-defined differences are receiving attention, and that when an animal's inheritance is being considered as a whole, simple Mendelism cannot provide adequate guidance for breeding operations. Also, the chromosome or gene theory in terms of which Mendel's results can be explained, is not the only possible interpretation of the known facts of inheritance. These more complex problems of heredity will be discussed in Chapter X, but first it is essential to describe the Mendelian theory of genetics before reviewing the facts discovered by scientists who use, and express their results in terms of, this theory.

The essence of the Mendelian theory (in its modernised form) is that a pup gets from each parent a certain collection of 'genes'. These genes are arranged in groups, usually likened to strings of beads, each string being called a chromosome. The chromosomes are present in the central part, or nucleus, of the cells of the body, and the number of chromosomes present in each nucleus is characteristic of the species, e.g. dogs have 78, human beings have 48, pigs have 38 or 40. In the ordinary cells the chromosomes are paired, two of each kind, so that it is better to say that the cells of a dog contain 39 chromosome pairs. When the reproductive cells are formed the pairs separate so that each sperm or ovum contains only one of each pair of chromosomes; when a sperm fertilises an ovum by uniting with it, the number of chromosomes is restored to 78, the offspring receiving one chromosome of each pair from each parent.

The genes are believed to be arranged in a definite order on the chromosomes, and genes situated in the same place on corresponding chromosomes all affect the same characteristic, but they may affect it in different ways, in which case they are said to be 'allelic' to each other. Thus several workers have described a gene known by the symbol b which tends to make the dog produce chocolate-brown pigment instead of black pigment. If its allele B is present, black pigment can be formed. There may be more than two alleles of the same gene but only two of an 'allelic series' can be present in any particular dog. The genes situated at different places on the same chromosome are said to be linked and do not necessarily all affect the same part or feature of the dog.

When a dog has two different alleles at a certain place on corresponding chromosomes, it is said to be heterozygous for those genes; if the two genes of a pair are of the same kind, it is said to be homozygous for them. Two other technical words must be defined: the 'genotype' of a dog means its gene-complex or total collection of genes, whilst the 'phenotype' is the dog itself, i.e. the outcome of its whole development, including the effects of the environment; where only one or a few characters are being discussed these terms are often applied in a restricted sense, meaning the genotype or phenotype in respect to those particular characters only.

It is now recognised that the simple idea of each gene having one effect, e.g. 'producing' a black coat-colour, is erroneous. The effectiveness of any one gene depends on the whole inherited make-up of the animal (including what is inherited in the rest of the sex cell besides the chromosomes) and on the opportunities for development provided by the animal's environment. The gene is regarded as a chemical substance which, given a suitable biochemical background on which to work, will tend to give rise to a series of complicated chemical changes leading to a certain result, e.g. the production of black pigment. This result can, however, be achieved only if the right chemical substances are present at every stage and if the general development of the animal proceeds normally. If another gene is present which prevents the development of hair, or if one is present which causes black pigment to be changed by oxidation to yellow pigment, then the gene B cannot in fact produce black hair colour. The gene is however still present in the dog, including its sex cells, and if, when reproduction takes place, the gene B escapes the company of the other two, then a pup with black coat colour may result. The final appearance of the dog is therefore the outcome of a kind of running battle between its various genes, but in a given environment the outcome of this battle is fairly constant for any particular collection of genes. The result is often a blend of the characteristics of the parents, but when a certain gene is known to be able to produce its full effect even in the presence of rival genes, it is said to be 'dominant' if the rival is its allele, 'epistatic' if the rival is not its allele.

Much confusion has arisen in scientific literature, and still more in 'popular' accounts of canine genetics, through failure to distinguish between true dominance and epistasis, and through attempts to treat all genes as simple dominant/recessive pairs. It must be clearly understood that the dog can only carry two of each allele (one on each of the paired chromosomes), but where epistasis is involved there may be many such gene pairs interacting together, and the dominance relation of a particular gene pair may itself be upset. Also, it is by no means necessary for one or other of a pair of alleles to be dominant: they may produce an intermediate effect, or supplement each other, or produce together an effect quite different from that produced by either of them separately. A similar variety of results may be the outcome of the combined effects of a number of non-allelic genes, epistasis being relatively rare. Where many genes are required to produce a certain effect, or where their effect is cumulative, they are called multiple genes, or sometimes polygenes.

Another cause of confusion is the apparent failure of some workers to realise that if the Mendelian theory is correct then (with rare exceptions) two members of every allelic series must be present in every dog.

In female mammals one pair of chromosomes can be distinguished (by its reaction to certain dyes under certain circumstances) from the other pairs, and is known as the pair of X chromosomes. The male has only one X chromosome, its partner being of a different kind, known as the Y chromosome. The male reproductive cells (spermatozoa) contain either a Y chromosome or an X chromosome but not both; the female reproductive cells (ova) each contain one X chromosome. This may be fertilised either by a Y-bearing sperm, making the offspring XY (male) or by an X-bearing sperm, making the offspring XX (female). Genes situated on these 'sex' chromosomes are called sex-linked genes.

Although Mendel himself did not know anything about the way chromosomes behave at cell division, the 'laws' which he deduced from the behaviour of his experimental peas fit in exactly with the behaviour of the chromosome.

He stated that the inherited 'factors' which give rise to different 'characters' in the parents remain separate in the fertilised egg, and are distributed independently of each other to the next generation of offspring.

Relatively few characters clearly show the typical segregation and re-assortment in inheritance which Mendel described. However nearly all published research work on dogs has been mainly concerned with inheritance of this rather unusually simple type. The information derived from these studies is extremely useful in certain limited circumstances, namely when a breeder wishes to introduce one or more of these characters into his strain (or to eliminate them) with a minimum of effort, or to establish a strain which will breed true or nearly true for one of these characters.

In the method of analysing inheritance by crossing individuals with contrasting characteristics, certain mating systems which were used by Mendel have become classical tests of the mode of inheritance. These matings are as follows:

The contrasted parents or P_1 generation are mated together to produce an F_1; these are then mated to each other to produce an F_2 in which the various parental characters sort themselves out independently of each other (unless 'linkage' occurs). The best known example of this is the cross between an individual 'pure' for a certain gene, i.e. carrying two of the same gene, and another individual 'pure' for its recessive allele. This cross leads to the famous 3 to 1 ratio, as illustrated in Diagram 1.

When several such gene pairs are involved in a cross the possible permutations and combinations become as complicated as a football pool coupon, and the mathematical principles involved are much the same.

DIAGRAM 1

Using the genes D and d, giving black or 'blue' coat colour

P_1	Black Dog	×	Blue Bitch	
genes	DD		dd	give:
F_1		All black puppies (mated together)		
genes		Dd × Dd		give:
F_2	1 'Pure' black	2 Black carrying blue	1 Blue	
genes	DD	Dd Dd	dd	
	('Homozygous dominant')	('Heterozygous')	('Homozygous recessive')	

When two independently assorting gene pairs are involved we get four types in the F_2 in the 'dihybrid ratio' 9:3:3:1, whilst with three gene pairs we get the 'trihybrid ratio', with eight different types in the proportions 27:9:9:9:3:3:3:1. Examples of such crosses and further explanations will be found in any textbook on Mendelian genetics (Lush, 1945; Whitney, 1948; Winge, 1950); the assortment of different types results simply from the fact of gene interaction described above. Thus the gene d causes clumping of the pigment granules in the hair (Burns, 1943) causing a black dog to appear smoky grey ('blue') and a liver dog to appear the colour known in Poodles as 'apricot' (known in Bedlington Terriers as 'liver'). If, in the example above, the blue P_1 bitch had also been pure for liver (bbdd) the F_1 would still have been black (BbDd) but in the F_2 one in four of the BB dogs would have been blue (BBdd) and one in four of the liver dogs would have been apricot giving the dihybrid ratio of 9 black (BBDd or BbDd or BbDD or BBDD), three blue (BBdd or Bbdd), three liver (bbDd or bbDD) and one apricot (bbdd).

The other classical test mating is called the backcross, which means the mating of any of the crossbred progeny (F_1 or F_2) to one of the parents or to any dog of either parental breed. The most useful and usual form of this is to backcross an F_1 animal to the parent showing the recessive characteristic: the resulting progeny should consist of approximately equal numbers showing

PLATE I

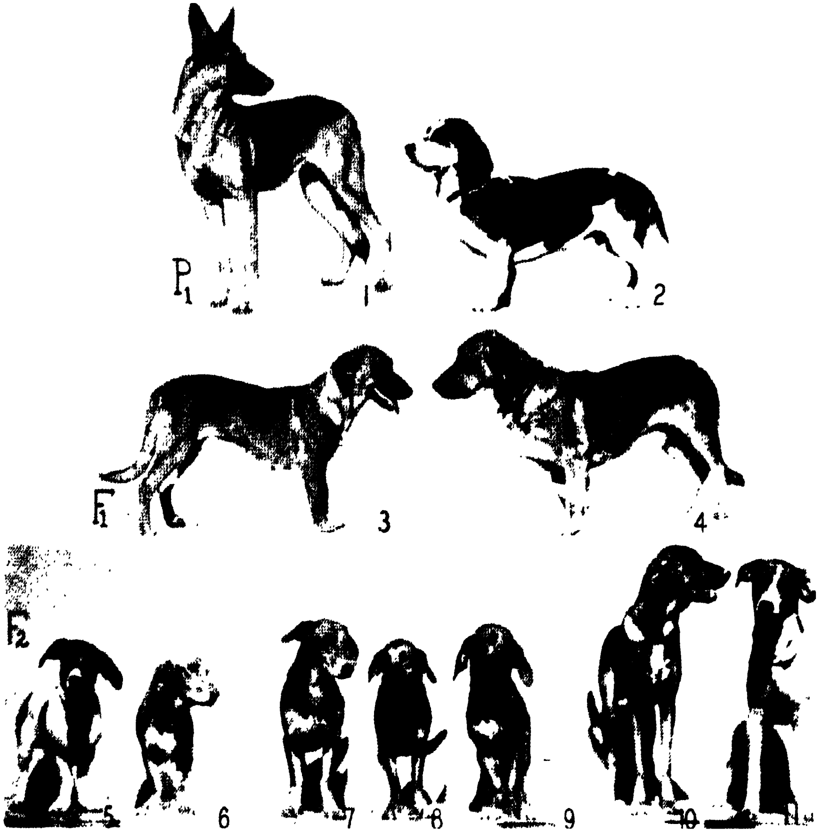


PLATE I. The Breeding of an F_1 and F_2. Cross between the long legged German Shepherd and the achondroplasic short legged Bassethound showing inheritance of leg length as well as other physical characters in the first and second hybrid generations. 1. German Shepherd ♀. 2. Bassethound ♂. 3-4. F_1 Brother (R.) and Sister (L.). 5-11. One litter of seven F_2 hybrids, all ♂♂ except No. 8.

♀ = female; ♂ = male

PLATE II (*a*) (*b*)

PLATE II. Contrasts in Size. (*a*) The Irish Wolfhound stands over 36 inches at the shoulders; (*b*) the Italian Greyhound (L.) only about 10 inches. (*c*) The Greyhound represents normal canine height, around 24 inches.

(*c*)

the 'dominant' and 'recessive' effects, if the difference between the P_1 parents was due to a single pair of allelic genes, one dominant over the other (Diagram 2).

If this does not happen some other type of inheritance must be involved.

DIAGRAM 2—BACKCROSS

P_1 DD × dd give:
F_1 Dd × dd give:
Black Blue
Dd and dd

The ratios actually obtained in breeding experiments are seldom exactly the same as those stated, especially if only a small number of puppies is bred. The theory is based on the assumption that fertilisation is 'random', i.e. that any sperm has an equal chance of fertilising any egg-cell. If this is true, statistical calculations show that exact agreement with the theoretical ratio is not to be expected in every litter, but is approached when a large number of puppies is bred. Thus if it is desired to find out whether a dog showing a dominant characteristic does or does not carry the recessive allele, it may be tested by mating it to a pure (homozygous) recessive bitch (as is done in test-mating Irish Red Setter dogs for 'night-blindness'; *see p.* 35). In the resulting litter, if the sire carries the recessive gene, the expected ratio is 1:1, i.e. equal numbers of pups showing the dominant and the recessive character. However, a litter of two pups, both showing the dominant feature, will not prove that the dog does not carry the recessive gene: out of every 4 litters of two pups, one litter will probably consist of two unaffected pups even if the dog is a 'carrier'. If six puppies are bred, the chance of none of the six showing the recessive quality if the sire is a carrier is only 1 in 64, i.e. only one in 64 litters of six will fail to reveal that the dog is heterozygous. This is the basis of the British Kennel Club's ruling that at least six pups must be bred from a test-mating, and none of them show retinal atrophy, before the dog or bitch being tested is passed as clear of the disease (*see also* Chapter IV).

Sometimes certain genes are described as 'modifying' genes because they appear to act only in the presence of some other particular gene, modifying its effect. However, they are really only demonstrating, in a particularly obvious way, that all genes affect each other's actions to a greater or less degree. Thus the gene R causes roaning of the white parts of certain dogs, its allele r permitting the white areas to be pure white or with only a few pigmented dots. It could therefore be described as a 'modifier' of the white-spotting gene s or simply as an epistatic to the 'solid colour' gene S.

It has been mentioned that more than two alleles of a certain gene may exist, and Diagram 3 illustrates the results of crossing dogs bearing different members of such an 'allelic series'.

Diagram 3

Genes	A	Black	in this order of dominance
	a^y	sable (dominant yellow)	
	a^g	wolf-grey	
	a^t	bicolour (e.g. black-and-tan)	

A dog may either carry two of the same allele, e.g. AA or a^ta^t etc., or it may carry two different ones, e.g. Aa^y, A^ga^t, etc. A cross may possibly involve as many as four different alleles

P_1	Black carrying sable	×	Grey carrying bicolour		
genes	Aa^y		a^ga^t		give:
F_1	Black	Black	Sable	Sable	
genes	Aa^g	Aa^t	a^ya^g	a^ya^t	

Two colours, four 'genotypes'. Thus a colour not seen in either parent appears in the F_1, and another—bicolour—can appear in the F_2 if the black carrying bicolour (Aa^t) happens to be mated to the sable carrying bicolour (a^ya^t).

	Aa^t	×	a^ya^t	gives:
	Black	Sable	Bicolour	
genes	Aa^y Aa^t	a^ya^t	a^ta^t	

Such variations occurring in the F_1 suggest that an allelomorphic series may be involved, but they can also occur due to the interactions of a number of independent gene pairs. Only further breeding tests can determine which type of inheritance is involved, and these tests are not simple or easy to apply, because of the large number of different gene combinations which are possible.

The test for whether or not genes belong to the same allelic series consists of breeding a series of F_1s and F_2s each involving only two of the suspected alleles, but in such a way that they are all proved directly or indirectly to be allelic with each other, i.e. to give a 3:1 ratio in the F_2 (or 1:2:1 ratio if dominance does not occur). Thus if the matings $a^ya^y \times Aa^g$ and $a^ya^y \times a^ta^t$ both give the 3:1 F_2 ratio, then a^y, a^g and a^t must be allelic; if AA and a^ta^t also gives the same F_2 ratio A must therefore be allelic with a^y and a^g as well as with a^t. Unfortunately many workers have asserted genes to be allelic without giving evidence of having conducted the necessary series of crosses.

If, when giving such symbolised formulae of the genes carried by a dog, it is not known whether an animal showing the dominant characteristic is homozygous or heterozygous, a 'dash' is used in place of the second gene of the pair. Thus a dog given as B– may be BB or Bb, and one given as a^y– may be a^ya^y, a^ya^g, or a^ya^t, but not a^yA.

In many cases scientists have failed to determine the exact mode of inheritance of complicated characters, or they disagree with each other's results. It is only in relatively simple cases that Mendelism gives a clear-cut answer without more prolonged breeding experiments than the ordinary dog breeder can attempt. If the breeding of an F_1, F_2 and a backcross does not make the situation clear to the breeder, either professional advice should be sought, or less precise methods of improvement, such as grading up or linebreeding, should be used.

The greatest use of simple Mendelism to the practical dog breeder is in applying knowledge which professional scientists have acquired, rather than in themselves determining which characteristics are inherited in a simple Mendelian manner and which are not.

The inheritance of the majority of the most essential breed characteristics depends on so many factors that the simple Laws of Mendel give no assistance to the breeder. In such matters as type, body size, temperament, fertility, hardiness and the finer shades of difference in such things as coat texture or colour, the scientist at present can only give general advice which is for the most part the same as would be given by any experienced breeder. Only two accounts of scientific attempts to establish outstanding strains of dogs have been published (Humphrey and Warner, 1934; Kelley, 1947), and one breed, Whitney's Bullhound, has been created by a scientist (Whitney, 1948). These will be discussed in a later chapter, as will other types of inheritance and other interpretations of the known facts, including the vexed question of the so-called inheritance of 'acquired characters'. Before attempting to discuss these wider aspects of genetics the results of their researches, as presented by scientists, will be summarised in the following six chapters. In these chapters the terminology used in summarising their work, is that used by the original authors themselves. Unfortunately in many instances scientific terms have been used very loosely. Thus many authors use the word 'dominant' to describe any character which is manifested in the F_1 of a cross, although on the evidence they present this result could equally well be due to epistasis or to gene interaction, and not to true dominance; in fact, the word 'dominant' is often used in the sense of 'prepotent' rather than in the strict scientific sense. Much confusion would have been avoided had the term 'dominant' been used only when the relation between two allelic genes was being discussed, the word 'prepotent' being used when the genetic cause of the apparent dominance of one type over the other was not known; future workers on canine genetics could with advantage adopt this course.

II

REPRODUCTION

VARIOUS conflicting estimates have been given of the number of chromosomes in domestic dogs, but the question seems to have been settled by Ahmed (1941) who found the diploid number to be 78, thus confirming the earlier work of Minouchi (1928). As is usual in mammals, the female has a pair of X chromosomes whilst the male has one X and one Y chromosome. Ahmed found minor differences between the chromosomes of a Sealyham Terrier and those of a Spaniel and a Spaniel cross. Future work may reveal other breed differences in chromosome structure and behaviour, but it is not very likely that the chromosome number varies between breeds; the different estimates of earlier workers were doubtless due to technical difficulties in rendering the chromosomes visible.

The frequency and duration of the oestrus ('heat') periods vary considerably from one bitch to another, and breed differences also undoubtedly exist. Most Racing Greyhound bitches come into oestrus only once each year, and this was true also of the Basenji when first brought to Britain. In the latter breed, however, heats are now occurring at intervals of six to eight months in many bitches in this country as is the general rule in other breeds. Iljin (1941) reports that the majority of F_1 and F_2 wolf × dog females come in heat once a year in winter, thus resembling the female wolf.

The occurrence of oestrus is influenced by environmental circumstances, especially by the periods of lengthening or shortening daylight (i.e. spring and autumn) which tend to produce oestrus. Kelley (1951) records that of 47 fertile heats observed (Sydney latitude) 16 occurred in autumn, 12 each in spring and summer, and 7 in winter.

Whitney (1927) found, from American records, that the greatest number of bitches were on heat in April, the smallest number in

December, and that no difference in this respect was shown by bitches in the southern States. This information was deduced by Whitney from the birth-dates of puppies, but since breeders do not usually have their bitches mated in the autumn and winter months, the data are incomplete. It would appear from the graphs given that almost as many bitches were in oestrus in February as in April. No breed differences are indicated. Hancock and Rowlands (1949) found the peak month for oestrus was July. Whilst no exact data are available, it is common experience that the presence of other bitches in oestrus seems to stimulate the onset of heat in bitches approaching their time but not yet due. Whitney (1946) found that when ovaries were transplanted from one bitch to another whose own ovaries had been removed, the oestrus periods of the latter bitch continued the cycles of the one from which the ovaries had been taken. Koch (1935) includes 'heat weakness', and hypertrophy of the vaginal mucus membrane leading to prolapse during oestrus, among conditions caused by hereditary abnormality of the anterior pituitary gland.

Studies on fertility have indicated that many factors affect litter size. Whitney considers that ovulation usually occurs on the 16th or 17th day of the heat period (about the 5th day of oestrus) and that matings about the 15th day are the most fertile. Kelley (1947) advises mating on the first day of acceptance by the bitch (when ovulation is considered to take place), and thereafter on alternate days until acceptance ceases, in case ovulation is continuous or delayed. Hancock and Rowlands found maximum fertility resulted from mating in the first four days of oestrus and a second mating 48 hours later. Spreull (1949) points out that it is inadvisable to allow a repeat mating more than two days after the first as puppies of different ages may result, leading to some being born prematurely and imperfectly developed.

Litter size shows strong correlation with breed size, the larger breeds having larger litters. Several interesting studies on this matter have been made, the most notable being those of Heape (1908), Druckseis (1935), and Whitney (1927), all of which deal also with the sex-ratio.

The sex-ratio (number of males per 100 females born) is higher in large breeds than in small ones and large breeds also have larger litters; this suggests that the smallness of the litters produced by

(*a*)

PLATE III. Litter Size. Litter size is correlated with body size, the average number of pups in a litter being greater in big than in small breeds. (*a*) Great Dane with ten pups. (*b*) Norwich Terrier with four pups.

(*b*)

smaller breeds may be due to overcrowding in the uterus with differential mortality of male embryos, rather than to the production of a smaller number of ova. Winzenburger (1936) found, however, that in Alsatians the sex-ratio decreased with increasing litter size. Heape notes that in the records which he used Greyhounds showed a higher sex-ratio in litters born between October and December than at other times of year, but litter size is not discussed in this connexion. Whitney found the sex-ratio averaged 143 in the colder months and 116.1 in warmer months.

TABLE I

Litter Size and Sex-Ratio

Size	*Breed*	*Average Litter Size*	*Sex Ratio*	*Reference*
Large	Bloodhounds	10·06	126·76	Heape, 1908
	Mastiffs	7·67	107·55	,, ,,
	St. Bernards	8·53	114·8	,, ,,
	Newfoundlands	8·45	119·13	,, ,,
	Alsatians	4 to 10 (83%)	110·45	Druckseis, 1935
		—	106	Whitney, 1948
		7·15	105·5	Winzenburger, 1936
	Border Collies	5·7	84·9	Kelley, 1951
	Collies and Sheepdogs	7·39	118·19	Heape, 1908
	Setters	8·68	96·43	,, ,,
	Retrievers	7·7	125·8	,, ,,
	Greyhounds	—	111·1 to 121·17	,, ,,
		6·5	—	Burns, 1943
Medium	Airedales	6·74	120	Heape, 1908
	Spaniels	6·77	117·37	,, ,,
	Bulldogs	6·32	126·82	,, ,,
	Bull Terriers	6·34	113·98	,, ,,
		—	127·0	Briggs and Kaliss, 1942
	Schnauzers	—	102·0	Druckseis, 1935
	French Bulldog	—	101·67	,, ,,
Small	Terriers	4·38 to 6·13	96·15 to 135·9	Heape, 1908
Toy	Toy Breeds	4·24	125·64	,, ,,
Breeds not stated, uniform environment		—	124·31	Whitney, 1939

The main data on these subjects are summarised in Table I. The sex-ratio of still-born pups is higher than that of live pups (Druckseis, 1935), i.e. more male than female pups die at or just before birth. Hancock and Rowlands (1949) found that the largest litters were born in autumn and winter, but the survival rate was lower than at other times of year; they suggest that this may be due to the larger litter size. Druckseis states that still births are lowest in the litters of 7 to 10 pups, but does not deal with subsequent survival rates.

Whitney (1948) suggests that the preponderance of males is due to differential fertility, possibly due to greater activity of the Y-bearing sperm, or to selectivity by the ova. The sex-ratio must usually be higher at fertilisation than at birth, since there is evidence that more male than female pups are still-born.

Some bitches are considered by breeders habitually to produce an excess of one sex, and this may be a factor in producing the excess of litters with only one sex noted by Winzenburger (1936), who however attributes it to monovular twinning. Whitney (1948) advances evidence that monovular twinning does occur in dogs, and Duke (1946) confirms this by describing two embryos with common chorion and yolk sac, which he observed. Some individual bitches have a characteristic high or low fertility. Whitney (1948) gives some interesting examples of this.

Several observers have confirmed the widely-held belief that pregnancy tends to be longer for male embryos (or predominantly male litters) than for females (Heape, 1908; Whitney, 1948), and the gestation period of Airedale Terriers was found to be significantly shorter than that of Foxhounds and mongrels (Hancock and Rowlands, 1949).

Freak (1948) mentions several whelping troubles which commonly occur in different breeds. Thus, the Dachshund is particularly subject to primary inertia followed by rapid general peritonitis; the Racing Greyhound frequently shows arrested foetal development and death of embryos at all stages, possibly due to genetic lethal factors; the Cocker Spaniel often fails to develop the early maternal instincts, hysterical and neurotic types appearing to be afraid of their new-born pups and refusing to 'mother' them. In other breeds whelping troubles result from structural abnormalities deliberately encouraged by breeders (Scottish Terriers, Bulldogs), or to a wide range of size variation within the breed leading to pups being oversized relative to the size of their dam (Sealyham, Scottish and Yorkshire Terriers, Corgi, King Charles Spaniel).

In some of Stockard's crosses bitches with abnormal maternal behaviour occurred (1941). Apgar (1940) studied the behaviour of bitches of various breeds and crosses during, and for a few hours after, whelping. He remarks on the 'disastrous awkwardness' of Great Dane-St. Bernard crosses, and states that leg-length plays an important role in postpartum adaptations of the bitch.

Abnormal maternal behaviour with a first litter does not necessarily imply that the bitch will behave abnormally on subsequent occasions; nor is abnormal behaviour always due to hereditary causes, since it can be the result of illness, fright or other temporary disturbance.

The subject of cryptorchidism is one which has received a great deal of attention from dog breeders in Germany and, to a lesser extent, in America. Fanciers of breeds of German origin in Britain and elsewhere tend to be influenced by the German attitude to this matter. The condition has been declared hereditary in many instances where the evidence was quite inadequate to prove anything. Continental judges have been known to condemn a dog as cryptorchid or monorchid when it was in fact perfectly normal. Cryptorchidism is a form of delayed or arrested development, and it is reasonable to suppose that it can be caused by conditions operating only during pregnancy as well as by hereditary factors. There is assuredly strong evidence that the condition 'runs in families' (Whitney, 1948), but inheritance is irregular and uncertain. Koch (1935) states that cryptorchidism is common in dwarf breeds and in breeds with short skulls (Bulldog types), and that it is inherited and recessive to the normal condition, but gives no evidence. He suggests that failure of the testes to descend is due to defective functioning of the anterior pituitary gland. Cryptorchids are sterile because the sperm cannot develop at the temperature within the body, but require the lower temperature of the scrotum. There is therefore no sense in discriminating against cryptorchids in the show ring, since even if their condition is hereditary they cannot pass it on. Dogs with one testis in the scrotum and one undescended are fully fertile but seldom sire cryptorchid pups. Such dogs are commonly called 'monorchid' but this term really means having only one testis, a different condition. The only real objections to cryptorchid dogs are advanced by Whitney (1948) who states that testis retained within the body have a strong tendency to neoplasms, and that such dogs are often of uncertain disposition. Neither of these statements is substantiated by other authors but, if necessary, both objections can be overcome by operative removal of the concealed testis.

Examination of such scientific information as is available on the subject suggests that it would be more logical to refrain from breeding from the dam of cryptorchids, than from the cryptorchids themselves; but the mode of inheritance is so uncertain and the operative treatment so simple that cryptorchidism would appear to be one of those abnormal conditions which, although partly hereditary in origin, are not worth the effort and sacrifice involved in genetic methods of elimination. The evidence does not support Whitney's view that the condition can be easily 'bred out', and there is no indication that cryptorchidism is any more frequent in breeds in which the matter has been totally ignored than in those breeds in which it has received so much attention (Bayer, 1936; Härtl, 1938; Koch, 1935, 1938; Whitney, 1948).

III

CONFORMATION, TYPE AND SIZE

Differences in conformation largely reflect differences in the underlying bony skeleton of the dog, although minor variations may be due to differences in muscling, or in the relative size and amount of folding in the skin. Conformation is of course of prime importance in all dogs kept for show purposes, since the 'show points' of any breed are largely concerned with the general make and shape of the animal. Its importance in animals kept purely for work is more debatable, and such research as has been done on this subject shows little direct relation between conformation and working ability. The majority of studies on the inheritance of conformation have been concerned with crosses between strongly contrasted breeds, and little is known of the genetics of those minor structural differences which are of such vital importance in show competition.

Marchlewski (1930) found that the 'greyhound-like' type of head in Pointers behaved as a dominant to the correct type of Pointer head, and states that this is true also of the lighter type of body structure. This is one of the very few observations on variations within a breed. The greyhound-like type, although stated to be 'distinctly dominant' to the normal Pointer type, is also stated to have been carried 'hidden' through several generations due to the presence of 'modifiers'. Thus it is not a clear case of simple Mendelian dominance.

Humphrey and Warner (1934) found that good development of chest and flank was closely related to health and efficiency in their German Shepherd Dogs, but that it was not strongly inherited. They conclude that 'more immediate results can be produced through exercise and proper nutrition during the development age' than by selective breeding for chest and flank development. They also noted that dogs which were reluctant to jump heights had less sloping shoulders than the willing jumpers had.

The most comprehensive study of the inheritance of form and skeletal type is that of Stockard and his collaborators (1941). This work involved many crosses, F_2s, and backcrosses between widely different breeds. These crosses showed a tendency for the F_1 to be intermediate in the majority of characteristics, and a single backcross of the F_1 to either parental breed gave, in almost all crosses, progeny resembling that breed to a remarkable degree, except where one parental type was Bulldog or French Bulldog. This suggests that a cross with another breed can be very quickly absorbed into a strain and will be difficult to detect after only one or two backcrosses have been bred.

The British K.C. ruling is that four such backcrosses must be made before the descendants of a breed cross can be registered in the appropriate breed register; in the case of an interbred, i.e. a cross between two varieties of the same breed, only three backcrosses are required.

Crosses between short-legged breeds such as the Dachshund, Basset Hound, etc., and breeds with normal legs, e.g. Alsatians, gave intermediate leg length in the F_1 and fairly clear-cut segregation in the F_2 into three leg lengths, long, intermediate and short. The inheritance of leg length was, however, complicated by the fact that the gene for short legs apparently affects heavy bone types more than light bone types. Saluki bones, described as 'slender but of dense hardness' tended to be dominant to Basset type; Bulldog bone was also dominant to hound bone, and Peke bone type dominant to Saluki bone. Anyone seriously contemplating crosses between breeds with contrasted structure should study Stockard's work for further details of his results. For the ordinary breeder the following information may be considered important: the achondroplastic short-leg gene affects heavy bone more than fine bone, and it will therefore be more difficult to get a short-legged dog with straight legs if the bone is heavy than if it is light. Light bone tends to be more dense and hard than does heavy bone (it may, however, be more brittle; the greyhound breeds seem to be particularly liable to fractures of the limb bones). In crosses between different bone-types the extremes (heavy and fine) tend to be dominant to intermediate types.

The animals used in Stockard's experiments were kept under carefully controlled conditions of diet, etc., in order to reduce variation due to environment to a minimum. It is well known, however, that the quality of the bone can be greatly affected by diet and exercise during puppyhood, heavy bone being the product of heavy feeding and possibly restricted exercise. The changes in proportion undergone by a puppy during growth are regulated only partly by its breed, although older-established breeds are less readily affected by environment than are those of more recent and mixed origin. High feeding during the first three or four months of life tends to produce heavy bone, large feet, a long back, and relatively short legs and neck, i.e. development comparable to that of early-maturing sheep (Hammond, 1932). Moderate feeding up to five or six months old followed by a period of high nutrition tends to thicken the long bones of the legs and also to coarsen the skull. In a breed such as the English Cocker Spaniel where the demand is for short backs, heavy bone, and fine long skulls the growth period of each part has to be watched and the diet nicely adjusted to encourage the development of these mutually antagonistic show points. Over-exercise is said to cause undue lengthening of the legs of Bulldogs, and rickets can produce short bent legs simulating the appearance of some of the achondroplastic breeds and their crosses. No scientific studies of the influence of nutrition on the conformation of dogs have been reported.

Whitney (1948) states, in opposition to Stockard's results, that crosses between short- and long-legged breeds give an F_1 of intermediate leg length, with 'a range in the backcross and no clear-cut segregation'. He worked with breeds as similar as possible in other respects, e.g. Basset × Bloodhound. However, a '¾ length' leg in hounds he found to depend on a single dominant gene. He also reports a recessive short-leg mutant in Cocker Spaniels. He concludes that usually short leg is dominant over longer types.

Sommer (1931) wrote an interesting paper on the changes in bone size due to age. The breeds studied were the Alsatian, Great Dane, Borzoi and Dachshund, and he found that the growth in length of the vertebral column was similar in all those breeds. He therefore expressed the measurements of other bones as a percentage of the length of the vertebral column, so that adjustment was made for the difference in size of the four breeds. The author is mainly interested in changes taking place between weaning age (eight weeks) and maturity, and glosses over the breed differences which are, however, obvious from the table which he presents. This shows that there are considerable differences between the breeds, both at birth and later. These are particularly noticeable in the case of the Dachshund, which, compared with the other breeds, is relatively higher in all skull measurements at all ages, but less so at maturity than at birth; all bones of the forelimb, including the shoulder blade, are long at birth but short in the adult Dachshund; the same is true of the hind limb. The pelvic breadth and width are high at birth but similar to the other breeds in the adult. Thus we have the rather remarkable discovery that, relative to the length of its spine, the new-born Dachshund puppy has a bigger skull and longer legs than has a new-born Alsatian, Dane or Borzoi, although, as is quite obvious, this is not true in the adult, in which the limb bones are disproportionately short.

Sommer's own conclusions do not appear to be justified by his data, for he states that by eight to twelve weeks of age the greater part of the relative skeletal growth has already been completed, and that all the breeds investigated show far-reaching agreement in the characteristic features. He is, however, correct in stating that in his dogs the skeletal growth was practically completed by nine months of age; that all measurements of the 'cerebral skull' (as distinct from the muzzle) show a marked relative decrease between birth and maturity, but the length of the nasal bone increases. This is obvious to any discerning breeder. The relative lengths of scapula, pelvis and humerus change little, but the lower limb bones all increase in relative length.

Konde (1947) describes a congenital subluxation of the coxofemoral joint in Alsatians, and expresses the view that these abnormal hip joints may be the result of breeding for long, low and extremely angulated hindquarters. Another type of hip trouble, known as Calvé-Perthe's disease, is stated to be common in the smaller terrier breeds, and to be inherited (Moltzen-Nielson, 1937). The exact mode of inheritance is not known.

The inheritance of skull shape was also studied in Stockard's experiments. Various short-faced breeds, such as Bulldogs, Pekingese and Griffons Bruxellois were crossed with breeds of normal head shape. It was found that whilst the results varied according to the particular breeds crossed, in general the short-faced type had a strong influence on the F_1 although it was not

PLATE IV

(*a*)

(*b*)

(*c*)

dominant. In most cases the F_1 pups were less uniform in skull type than in other characters such as length of leg. The length of the upper and lower jaws was inherited independently so that in these crosses, especially in the F_2s, many instances of 'overshot' and 'undershot' jaw occurred, sometimes in extreme degree. Backcrossing to the more normal-skulled parent gave uniformly normal-skulled litters, but backcrossing to Bulldog types gave wide variation in the puppies. The authors note that 'chondrodystrophy is commonly found among dwarf types, and overgrowth of skin is common among giant breeds', and there may be linkage between dwarf size and achondroplastic short legs. Some of their experimental dogs showed new combinations of these characters with most astonishing results. In the F_2 from the Basset and Bulldog cross both giants and dwarfs occurred, including one individual which could readily be mistaken for a well-bred smooth-coated St. Bernard. In general crosses involving Bulldog 'blood' tended to throw extremes of body size in the F_2 and backcross generations. Structural disharmonies of many kinds occurred, e.g. upper and lower jaws of unequal size, forelegs not in proportion to hind legs, eye socket too small for eye, etc.

Crossing Griffons Bruxellois with Dachshunds gave, in addition to various disharmonies and deformities in the F_2, hairlessness, undue susceptibility to mange infection, and a tendency to rickets.

The large skull size and cranial crest of the Basset were recessive to the small skull size and flat-topped cranium of the Dachshund. Wriedt (1929) found Peke shape of skull to be conditioned by a single partially dominant factor in crosses with the Dachshund. The F_1 skulls were intermediate but variable. He also found that short leg was incompletely dominant (Dachshund × Schnauzer backcrossed to Fox Terrier).

Klatt (1944), crossing Whippet and Bulldog, also found great variation in size in the backcross to Bulldog. The skulls were intermediate (F_1 skull nearer Whippet but heavy, with Bulldog

Plate IV. Contrasts in Head Shape. This varies in length, breadth, and the relative size and shape of skull and muzzle. The Cairn Terrier (*a*) represents the 'normal' head shape, not very different from that of the wild canidae, whilst the Bulldog (*b*) and the Borzoi (*c*) are opposite extremes.

breadth). Tooth size was variable and not correlated with body size. Bone porosity was less than in Bulldogs and the sutures were slow to disappear, as in Whippets.

Ritter (1937) also notes that tooth size and jaw size are inherited independently (Alsatian × Boxer). The F_1s were intermediate in conformation and skull shape but almost none were undershot. About 50% of the backcross to Boxer were undershot, i.e. the condition was incompletely recessive. Grüneberg and Lea (1940), in a paper of special interest because it concerns a jaw anomaly within a breed (Long-Haired Dachshund), describe a recessive gene whose primary effect is shortening of the lower jaw (mandible); this causes faulty occlusion of the front teeth in puppies, which apparently leads to lengthening of the upper jaw. The result is an overshot condition. The teeth of these overshot dogs are smaller than normal and it is suggested that this may cause shortening of the lower jaw but wider spacing of the teeth in the upper jaw. Kelley (1951) reports the occurrence of three Border Collie puppies with shortened lower jaws among twelve offspring of a dog and his two half-sisters, all the parents being normal. This suggests a similar recessive mode of inheritance. Gaspar (1930) presents data on crosses of the Ceylon Hairless Dog and the Dachshund. The former, in addition to lack of hair, is characterised by reduction of the number of teeth. In crosses this reduction, which is stated to be caused by the same factor as hairlessness, is incompletely dominant. The shape of different skull bones and parts of bones is inherited independently, depending on the effect on each centre of ossification. This leads to peculiar recombinations and abnormalities in crosses. Humphrey and Warner (1934) report that the normal number of teeth was dominant, in their dogs, to a reduced number; they considered that several different gene pairs or allelic series were concerned in the inheritance of tooth-number.

Phillips (1945) describes the occurrence of overshot jaw in a strain of Cocker Spaniels, and concludes that its irregular appearance in the strain can best be explained on the assumption that it is caused by a recessive gene with multiple modifying factors (this does not seem a very instructive conclusion!). Maximum shortening of the lower jaw appeared to be incompatible with life; affected pups often died young, and the litters containing them were usually small, and had an abnormal number of stillborn pups. Like other authors he notes that the overshot condition may not develop until after weaning. He expresses his belief that the occurrence of jaw abnormality in this strain is connected with the crossing of English and American Cockers.

Hauk (1941) found that down-face (i.e. convex profile of top of head and muzzle, as in Bull Terriers, etc.) was not dominant. Hall (1943) found that the skull of an F_1 coyote and Wire-Haired Fox Terrier resembled the coyote in those characters which were not intermediate.

Iljin (1941) crossed Alsatians with wolves and studied many attributes of the F_1 and F_2 descendants, making very detailed observations on the skulls. The F_1 was intermediate in the majority of skull characters, and segregation occurred in the F_2. Iljin was perhaps too ready to assume that the Alsatian skull is representative of dog skulls in general and to attribute everything to inheritance of the simplest Mendelian type. The detailed data, although very valuable in other connexions, are not of sufficient concern to dog breeders to warrant inclusion here. One general observation is of interest, namely that wolves reared in captivity are said to become shorter in the muzzle, and higher and broader in the skull, thus approaching the skull shape of the Alsatian. This suggests that the skulls of wild wolf-dog crosses should more nearly approach the wolf type than did those reared in captivity by Iljin. He also points out that, in regard to general conformation 'the majority of the characters met with in the wolf, when met with in dogs are considered as consequences of unfavourable environmental conditions, e.g. narrow chest, elbows pressed to the chest, and front feet turned out'.

Vau (1939) studied the length and direction of the bony passage of the outer ear (*meatus acusticus osseus*) and found these varied according to breed, being correlated with the position of the ear muscle; in adults it was independent of age and sex.

Koch (1935) mentions a narrowing and reduction of the larynx inherited as a recessive factor in Skye Terriers. Klatt (1939) crossed an apparently normal Whippet dog with a French Bulldog bitch showing abnormal thoracic vertebrae and a nodulated tail. Skeletal abnormalities occurred in 8 of 13 F_2 pups, and 7 of 9 backcrosses. The factorial basis of the anomaly is not clear, except that a fusion of the last rib with its vertebra may be due to a simple Mendelian recessive factor.

The inheritance of dew-claws has received attention from several authors. Stockard (1941) found that double dew-claws behaved as a simple recessive to single dew-claws, and Guerreiro (1947) stated that the five-toed and six-toed condition can be hereditarily transmitted, but presented no exact genetical data. Whitney (1948) concludes that a dominant gene is responsible for the occurrence of dew-claws on both hind legs. Keeler and Trimble (1938) report variable dominance of dew-claws in Dalmatian × Collie crosses, and Falaschini (1941) concludes, from three experimental matings between Brackes and Pointers, that the dew-claws on the hind legs of the Bracke are due to a dominant Mendelian factor. Seiferle (1927, 1928) discusses the occurrence of extra toes in various breeds, but no useful genetical data are presented. Kelley (1951) states that among his dogs dew-claws on the hind legs are certainly not due to a Mendelian dominant factor, since they have occurred on puppies neither of whose parents have dew-claws, and his data suggest a recessive mode of inheritance.

Apart from the inheritance of dew-claws there is little information on the genetics of foot characters, but in Pointers (Marchlewski, 1930) and in Alsatians (Humphrey and Warner, 1934) short, compact shape ('cat-foot') has been reported to be dominant to long, open-toed shape ('hare-foot'); Kelley's experience, however, does not support this view. Foot shape is, of course, very much affected by nutrition and exercise, etc. The Chesapeke Bay Retriever and the Newfoundland are reported to have webbed feet which assist them in swimming, but there is no information about its inheritance.

Several workers have attempted to solve the problem of the inheritance of short or 'natural docked' tails (see Table 2), and the general conclusion seems to be that short tail is dominant or incompletely dominant to normal tail length, but that the gene is lethal in the homozygous condition so that 'natural-docked' puppies which survive are always heterozygotes. Lienhart (1932) however found, in Breton Spaniels, that the 'tailless genes' had no lethal effects, and that the inheritance of taillessness was due to multiple factors, not to a single gene.

The kinky tail of Bulldogs and French Bulldogs has been reported by several authors to be dominant to normal straight tail, and Stockard *et al.* (1941) state that an independent pair of genes determines whether the tail shall be long (dominant) or short. Whitney (1948) however, found this explanation too simple. The latter author also found no simple Mendelian inheritance of tail carriage or length in crosses between breeds. The F_1 tails were usually intermediate between those of the parent types. Multiple factors are probably involved. This is supported by most of the crosses in Stockard's work.

The inheritance of ear size and carriage is of the greatest importance to breeders of show dogs, and is of interest even to many breeders of working dogs, since ear carriage is one of the few points of appearance which seem to be regarded as important in Border Collies, and also to some degree in working terriers, hounds and gun dogs. The texture of the ears, whether thick or thin, also receives some attention from breeders of hounds on account of its supposed connexion with the sense of smell, long ears of thin texture being widely believed to be correlated with good scenting powers.

The most comprehensive research on this subject was carried out by Iljin (1937) who postulates a series of three allelic genes: H^a (semi-erect 'terrier' ears); H (lop-ears, as in hounds); and h, erect. H^a being completely dominant to H and h, whilst H is only partially dominant to h, the heterozygote Hh having semi-erect ears. In addition an independent gene is stated to be responsible for the type of ear found in the Borzoi, and another type of lop ear, recessive to erect ear, also occurs. It seems probable that the effect of any factor for ear carriage will depend on the size and texture of the ears, and intermediate types of ear-carriage, between those described by Iljin, also occur. Iljin's hypothesis thus oversimplifies the situation and multiple factors are undoubtedly involved, perhaps as modifiers of the genes named by him. Kelley (1951) states that among his dogs fully erect ears breed true. This type can be accepted as a simple recessive.

Marchlewski (1930) found the small ear size of the Alsatian dominant to the large ear size of the Pointer, and the small triangular ear of the English Pointer dominant to the larger ear of the German Pointer. Pendant ear carriage is imperfectly dominant to erect carriage in crosses between breeds, but within the Alsatian breed Humphrey and Warner (1934) found that erect ear seemed to be incompletely dominant to faulty (drop) ear carriage. Stockard (1941) concluded that the erect ear of the Alsatian was due to multiple factors and that in certain crosses ear carriage is independent of ear size. Pendant ears seem to dominate other types of ear carriage in crosses. They give illustrations of a dog produced by backcrossing an F_1 Boston Terrier × Dachshund to a Boston Terrier; this dog has the large ears of the Dachshund carried erect as in the Boston Terrier, producing a most surprising appearance.

To summarise the results of these numerous researches on the inheritance of conformation in dogs, it seems clear that only in rare cases can differences be attributed to simple, clear-cut Mendelian factors. In many instances the same result may be brought about by different causes, e.g. short legs may be due to a simple recessive gene, to partially dominant factors, to multiple factors not showing dominance, or even to rickets or to general poor nutrition. Similarly varying and conflicting results are reported for jaw anomalies, short-tailedness, skull shape, etc. The few clear statements which may be usefully borne in mind by breeders are as follows:

(1) Heavy bone is less hard and dense than light bone, and is more affected by inherited achondroplasia (and also probably by rickets).

(2) Crosses involving Bulldog or breeds derived partly from Bulldog are liable to produce extreme variation in size and various structural disharmonies. The Bulldog influence is always very manifest in the descendants of Bull-breed crosses.

(3) Crosses involving giant breeds are liable to produce excessive loose skin. Those involving dwarf (Toy) breeds with normal

Plate V. Contrasts in Ears and Skin Folds. The skin of the Bloodhound (*a*) is too big for it, especially around the head and neck, whilst the opposite extreme is represented by the Miniature Black-and-Tan Terrier (*b*) with its tight skin, 'clean' neck, and erect ears; the Great Dane (*c*) is intermediate in both ears and skin.

breeds are liable to produce abnormalities not obviously connected with size, such as hairlessness.

(4) Structural differences, if very marked, occurring suddenly within a breed are quite likely to be inherited in a fairly simple manner.

(5) Factors which are harmless within a breed may become lethal when combined by crossing with other factors which accentuate them or conflict with them in development.

(6) The inheritance of ear and tail type and carriage is very complex, and the more extreme types, such as erect ears or tightly curled tails, will be difficult to restore if once lost from a strain.

(7) Dew-claws have a strong tendency to occur frequently among descendants of dogs with dew-claws, although simple Mendelian dominance is not always evident.

(8) There is no accurate scientific information on the inheritance of those important show points commonly supposed to be essential for 'soundness', such as straight fronts, tight elbows, slanted shoulders, well-bent stifles, and 'correct' movement. Wild canidae and working strains of dogs, although often 'unsound' by show standards, do not appear to be in any way incapacitated thereby.

PLATE VI. Contrasts in Types of Long Coat. There are several kinds, three of which are illustrated here: (*a*) silky and very fine as in the Yorkshire Terrier; (*b*) straight outer coat with fine woolly undercoat as in the Chow-Chow; (*c*) woolly and thick as in the Old English Sheepdog.

IV

PHYSIOLOGICAL PECULIARITIES, ABNORMALITIES AND DISEASE

IN THIS chapter we shall describe what is known of the inheritance of various abnormal physiological tendencies and conditions, and of a few anatomical peculiarities. It is not always easy to determine the point of division between the extreme of normal variation and the commencement of abnormality; such division is in fact inevitably arbitrary and merely a matter of convenience. Therefore skeletal abnormalities are included with other skeletal variations in Chapter III, abnormalities directly affecting reproduction in Chapter II, and abnormalities of behaviour, other than reproductive behaviour, in Chapter V.

Perhaps the most striking inherited physiological peculiarity is that of the Dalmatian breed, which is less able to break down uric acid into allantoin than are other breeds, and which consequently excretes a larger amount of uric acid in the urine. This peculiarity does not seem to have any serious effect on the animals, although Dalmatians are reported to be slightly more susceptible to kidney stones than are other breeds (Keeler, 1940). The characteristic 'high uric acid excretion' behaved as a simple Mendelian recessive in crosses between Dalmatians and other breeds (Trimble and Keeler, 1938) and segregated independently of the spotted coat colour characteristic of the Dalmatian. Dalmatians also have fewer intranuclear crystals in their liver-cells than have other breeds; this condition is inherited and appears to be associated with high uric acid excretion (Weatherford and Trimble, 1940).

Two papers (Hutt *et al.*, 1948; Merkens, 1938) have reported the existence of hereditary haemophilia in dogs, but the breed is not stated in either instance; the condition is due to a single recessive sex-linked factor, and consequently in the families reported only males exhibited the condition, the heterozygous females being apparently normal. No haemophilic pup survived beyond seven months of age, so that there was no possibility of breeding a homozygous haemophilic female, and the condition was passed on in the strain only by the heterozygous bitches. The symptoms were lameness, swellings due to internal bleeding, and failure of the blood to clot when open wounds occurred. By special methods of rearing, Brinkhous and Graham (1950) succeeded in raising haemophilic male dogs and, by breeding them to heterozygous females, obtained haemophilic females in approximately the expected numbers.

The existence of three different blood groups in dogs has been established but no information on their inheritance is yet available (Dahr, 1937; Lauer, 1930; Olson, 1940).

Although not strictly a physiological condition, it seems appropriate at this point to mention the remarkable study of Herman (1926) on the heart of the Racing Greyhound. The average weight of the heart in proportion to the body (expressed as heart weight in grams per kg. of body weight) was found to be higher in the Racing Greyhound than in any other animal in which it has been measured. The weight in Greyhounds ranged from 11·3g. in an untrained puppy to 17·3g. in an old racing dog; with an average of 13·4g. The average for 200 mongrels was 7·98g., with a maximum of 9·98g. The author concludes that Racing Greyhounds are endowed with a proportionately larger heart at birth and that this responds to training by enlarging even more. No studies on the inheritance of relative heart size have been reported.

Another condition affecting the blood vascular system is reported by Ollerhead (1939) in several closely related English Setters; the condition consisted of a tendency to haemorrhage and periodic diarrhoea, and was thought to be an allergic manifestation combined with abnormal permeability of the capillaries. Its exact mode of inheritance was not determined.

Despite the large amount of attention which research workers have devoted to problems connected with the endocrine glands, there is little genetical information concerning them, apart from the *magnum opus* of Stockard and his collaborators (1941). These workers found that the histological structure of the pituitary, thyroid and parathyroid glands appeared to be of greater significance than the size (whether absolute or relative to the body weight) of the glands. The size of the thyroid is very variable within breeds and is not correlated with body form; it appears to be inherited as a genetic character itself. The giant breeds have relatively smaller thyroids than have normal dogs.

The size of the pituitary varies less than that of the thyroid, and independently of thyroid size; it is greater in large than in small dogs, but small dogs have larger pituitaries in proportion to their body weight.

The structure of the thyroid appears to have considerable significance and to be correlated with breed type. Thus the thyroids of Bulldogs, French Bulldogs, Pekes and Boston Terriers are of one type (though not identical with each other), whilst dogs of more normal head-shape have a different thyroid structure. Of special interest are the thyroids of 'Toy' breeds such as the Griffon Bruxellois and the Maltese Terrier, which are like the thyroid glands of new-born puppies, suggesting that these breeds in certain ways 'never grow up'. Apparent histological hyperactivity of the thyroid gland was not always accompanied by physiological or nervous manifestations of hyperthyroidy. The pituitary glands also showed differences characteristic of breed types, and in particular, in the Bulldog types exhibited cysts and a peculiar cord-like arrangement of cells in the *pars distalis*.

Obouissier (1940) confirmed the existence of cysts and other abnormalities in the pituitary glands of short-faced breeds and their inheritance in crosses with breeds of normal long head shape.

Marchlewski (1930) suggests that over-activity of the thyroid gland may be associated with an inherited periodic eczema in the English Pointer. Unfortunately, neither he nor Stockard (1941) examined the thyroid glands of Pointers histologically.

Richter (1931) found no evidence that goitre was inherited by the descendants of two bitches manifesting this condition, but Brouwers (1950) suspected an hereditary origin in an affected Wire-Haired Fox Terrier bitch, some of whose offspring also had goitre.

Rickets is a condition which may be associated with abnormality of the parathyroid glands (Stockard, 1941). A strong tendency to develop rickets may be inherited, so that affected puppies develop rickets on a diet which would provide adequately for the bone development of normal pups. Thus Stockard (1941) found that among the descendants of crosses between Griffons Bruxellois and Dachshunds, some individuals, particularly in the F_2, developed rickets despite special dietary supplements of bone meal and vitamin D. Raghavachari (1943) found that a bitch, whose own blood serum was low in organic phosphorus, threw rickety pups by two different sires, one of which also sired rickety pups from another bitch; 16 other bitches, kept under the same conditions, all reared normal pups without rickets. It would therefore appear to be advisable to refrain from breeding dogs or bitches which produce rickety pups under conditions in which the offspring of other individuals develop normally. It may also be undesirable to provide too ample supplies of bone meal and vitamin D since this may conceal an undue tendency to rickets, or, on Lysenko's theory (see Chapter X) may even lead to an increased need for such supplements, i.e. to the development of rachitic tendencies in the strain. It should be noted that the conditions described by these authors as rickets might possibly be some other osteodystrophy and not true rickets.

The condition known as 'Scotch cramp' or recurrent tetany (Klarenbeek *et al*, 1942; Smythe, 1945) is also suggestive of parathyroid dysfunction, although it has not been attributed to this cause. Smythe reports its occurrence in a Cocker Spaniel bitch and a Wire-Haired Fox Terrier dog, as well as in Scottish Terriers (in which breed the condition is sufficiently common to have originated the term 'Scotch cramp'). The condition is more common in dogs than in bitches, and is seldom manifested before the dog is six months old, occasionally not until three years of age. Smythe considers the condition to be a recessive hereditary neurosis. Kollarits (1924) describes a somewhat similar condition in Airedale Terriers, consisting of an inherited permanent trembling, especially in the hindquarters and tail. He attributes this to 'inbreeding degeneration' but does not trace its exact mode of inheritance.

Iljin (1932) includes asthma and epilepsy as diseases, some forms of which are inherited. Epileptic tendencies are widespread in certain breeds, notably the Fox Terrier (both types), and appear to 'run in families'. Gray (1930) states that Toy Spaniel and Bulldog breeds are especially liable to spinal neuritis and spinal pachymeningitis followed by paralysis.

A nervous affliction of a different kind is described by Stockard

(1936, 1941) in crosses between Great Danes and St. Bernards. This consists of paralysis of certain muscles of the hind leg, resulting from the death of certain nerve-cells in the spine, and is found occasionally in the pure breeds concerned. Stockard considered the condition to be dependent on probably three partially dominant mutant genes, paralysis being more pronounced the more nearly homozygous the individual was for all three mutant genes. Unsoundness of the hindquarters is certainly extremely widespread in these two breeds, and apparently less so in a third giant breed, the Irish Wolfhound. Whether this unsoundness is due to a small number of specific genes or to more general causes is not definitely known, but the peculiar stilted action commonly seen in unsound Great Danes certainly conforms to Stockard's description of compensatory action of the muscles not affected by paralysis.

Phillips and Felton (1939) report that umbilical hernia is inherited as the operation of multiple recessive genes in Show Collies, Cocker Spaniels and Bull Terriers and is not associated with any other type of hernia nor linked with sex, colour or deafness.

Marchlewski (1930) and Koch (1935) both state that periodic eczema depends on inherited dominant factors probably associated with thyroid dysfunction. Witzigmann and Käb (1937) discuss breed differences in susceptibility to skin diseases; they state that in general wire-haired breeds are least susceptible, long-haired breeds are most susceptible, and short-haired breeds intermediate, but that certain breeds show a special susceptibility or resistance to certain skin diseases.

Koch (1935) considers that a number of congenital conditions such as cleft palate, corkscrew tail, oestrual weakness, oestrual prolapse, periodic eczema, etc., are all due to hereditary abnormality of the anterior pituitary rather than to a series of linked genes. Steiniger (1940-42) discusses hereditary hare-lip in the dog, but inheritance is doubtful.

The question of resistance or undue susceptibility to infectious disease is obviously of great importance to dog breeders. Although thousands of case histories are recorded annually by veterinary surgeons, the only attempt at statistical study of breed differences available is that of Whitney (1948). He noted the extreme susceptibility of Bloodhounds to death from distemper (this is well known to breeders, and is also mentioned by Smith (1939)), whilst his Bullhounds (descendants of crosses of Bull Terriers and Bloodhounds) showed a very low mortality in the same outbreaks of the disease. Moreover those Bullhounds which most resembled their Bloodhound ancestors in appearance were the most likely to die. The Bull Terrier as a breed appears to recover well from the disease. Another virus disease, which Whitney calls 'house-dog disease', was found seldom to be contracted by Scotties, while Alsatians and Dachshunds appeared to be especially susceptible; these two breeds were also the most likely to develop encephalitis in the later stages of the disease, whilst the lowest percentage of encephalitis among affected dogs occurred in the Boston Terrier and Setter. Susceptibility to fits during the first (high fever) stage of distemper was high in Bloodhounds and Beagles and low in Scotties and Cockers.

It should be remembered that Whitney was dealing with American Cockers. The English Cocker has the reputation of being excessively prone to fits, but no reliable statistics are available. Cairn Terriers and Scotties are reputed resistant to distemper, whilst the present Irish Setters in England are alleged to be second only to the Bloodhound in susceptibility. Resistance to paradistemper ('hard pad disease') very evidently varies greatly, but once again no statistics are available.

In a recent study of a spontaneous epidemic of viral hepatitis in a community of 38 dogs, Parry (1950) includes valuable information on the morbidity and mortality rates according to breeds. The four adult animals (Greyhound bitches) were unaffected, as were the three Smooth-Haired Terrier puppies. The greatest number of severe cases and the highest mortality rates occurred among the Greyhound puppies which showed signs of more severe disturbance of the digestive and cardiovascular systems than did the other breeds. All the pups which developed hysterical seizures were Irish Red Setters. Three of the four Border Collies developed the disease but none died. In each breed, some individuals showed no symptoms of the disease although fully exposed to infection.

Sometimes resistance to disease can be passed on by a bitch to her offspring through her blood stream or her milk, and sometimes diseases can be passed on in the same way. This is not 'true' inheritance in the geneticist's sense, but it may amount to very much the same thing as far as the breeder is concerned. Roundworm larvae are reported to be able to enter the puppies' blood from the mother during pregnancy, and follicular mange is said to be similarly 'inherited'; puppies whose dam has had and recovered from distemper are stated to show resistance to the disease as long as they are suckling.

Hereditary deafness has been reported in several breeds of dog and is often associated with white coat-colour. Thus at one time deafness was very common in white Bull Terriers but, according to Crew, this was due to the fact that a certain widely used Stud dog passed on a recessive gene for deafness to his numerous offspring: through later inbreeding many individuals homozygous for this gene, and consequently deaf, were produced. Certainly not all white Bull Terriers are necessarily deaf, but the white Collies which result from the homozygous condition of the factor, which in the heterozygous state produces merle coat-colour, are nearly always deaf, and partially or totally blind. The same condition occurs in the Norwegian Dunker breed, and in Harlequin Great Danes. It has been suggested that Harlequin is a modification of merle coat-colour which by selection has been established in a gene complex in which it is generally harmless; however, breeders do not usually mate two harlequins together so that the majority of this colour are not homozygous, and homozygotes are in fact defective (Whitney, 1948). Merle coat-colour occurs quite frequently in Great Danes but as this colour is not permitted in the show ring, merle puppies are usually destroyed at birth.

Dechambre (1932) describes a litter of Fox Terriers whose dam was deaf and white, but the sire was normal; all the bitch puppies were deaf but the dogs were all normal. Unfortunately all died of distemper before further breeding tests could be made. Pearson and Usher (1929) mention the occurrence of deafness and also weak eyesight among albinotic Pekingese, but Gray (1933) states that albino animals are seldom deaf. Kelley (1951) describes a form of deafness in Border Collies, which did not become noticeable until about two years of age. Inheritance seemed to be due to a single recessive factor, and was not associated with white coat colour.

Several hereditary abnormalities of the eye, leading to partial or total blindness, have been reported. The type which occurs in dogs homozygous for the merle colour factor is known as micropthalmia: the eyes are excessively small and the pupil often oblique, while pigment is reduced or absent. Heterozygous merle dogs often have 'wall' eyes but their structure and vision is normal. 'Wall' eye (affecting one or both eyes) can occur independently of merle coat-colour, when it does not behave as a dominant in inheritance. Linkage with the gene for merle has been suggested as a possible explanation, but in that case the merle gene must enable the gene for 'wall' eye to take effect when not homozygous, i.e. reverse its dominance.

Iljin (1932) lists as hereditary defects of the eye, glaucoma, coloboma, cataract and periodic inflammation of the eye, but gives no details of the mode of inheritance. Hippel (1930) describes a family of Alsatians in which cataract was inherited as a simple Mendelian dominant. Host and Sreinson (1936) report the occurrence of a form of cataract in Pointers in Scandinavia. The condition develops during the second or third year of life and leads rapidly to complete blindness. The tendency is hereditary and can be traced back for many generations to the bitch, Lady of Gammelgord, but the exact mode of inheritance is not known. Kittel (1931) discusses the occurrence and inheritance of dermoid cysts of the cornea, and of coloboma, in a family of St. Bernards, but the genetical data are very unsatisfactory. These defects are stated to be common in the St. Bernard and Newfoundland breeds. Whitney (1948) states that glaucoma 'runs in families' and is particularly common in Wire-Haired Fox Terriers, but that it is not inherited in any simple Mendelian manner.

Gray (1930) states that abnormal growth of eyelashes (trichiasis and distichiasis), so that they cause trouble by touching the eyeball, is common in Toy Spaniel and Bulldog-type breeds. In these breeds the prominence of the eyeball combined with low corneal sensitivity renders the eyes very susceptible to damage. An article in the *Veterinary Record* (1930) mentions the occurrence of inverted eyelids in Chows and Pekes; this condition occurs in other breeds, and is apparently inherited, but no exact data are available in the literature.

The most topical of hereditary eye afflictions at the present time is the condition popularly known as 'night blindness', more correctly named progressive retinal atrophy (Hodgman *et al.*, 1949). This condition was found to be widespread in the Irish Setter breed, particularly in the most famous show-winning strains in Great Britain. Breeding tests yielded strong evidence that the condition was inherited as a simple Mendelian recessive, and at the request of breeders the British Kennel Club took steps to prevent afflicted Setters and those known to be carriers from being registered or bred from, except for the purpose of test matings. Great sacrifices have been made by breeders in order to eliminate this defect from the breed, but occasional apparent exceptions to the general mode of inheritance have caused some breeders to question the soundness of the measures being taken. Statistical theory suggests that the test-mating procedure recommended by the Kennel Club will very occasionally fail to reveal that the animal being tested is a carrier, although the minimum number of pups required (six) will almost always be sufficient for the purpose. Also, breeders' diagnoses of whether the pups are affected or not may sometimes be erroneous, an error which could be avoided if the Kennel Club required that the puppies be examined by a veterinarian or other fully competent person. Despite these and other less probable eventualities which could lead to errors in test-mating, the method adopted has in fact led to a great reduction in the occurrence of retinal atrophy in the Irish Red Setter, and the intelligence and stamina of the breed appear to be improving incidentally, probably as a result of the outcrosses to less popular strains which breeders were forced to use. Thus progressive retinal atrophy in the Irish Red Setter constitutes one of the rather rare instances in which the use of genetical means of elimination was clearly indicated, and fully justified in practice.

Apparently similar retinal atrophy has been found in dogs of other breeds, but its inheritance in these breeds has not been established. If both hereditary and non-hereditary forms of the disease exist the identification, and therefore the elimination of the hereditary form, will obviously be most difficult, and this raises the whole complicated question of the value of genetical methods of eliminating defects and diseases. As with other inherited characteristics, what is inherited is not a defect or a disease, but a developmental tendency or susceptibility. If methods of environmental control can, with reasonable ease, prevent the tendency reaching fulfilment, then the inherited condition becomes unimportant from the breeder's point of view. Genetical methods of elimination are always costly and difficult, even where a genetical factor behaving in a simple Mendelian manner is involved. Where less clear-cut inheritance is manifest genetical methods are even more difficult and uncertain. In withholding an animal from breeding because it can pass on a known defective tendency, all the desirable qualities of the animal are also sacrificed. Therefore it may at times be desirable to breed from an outstanding animal especially if its good qualities are not widespread in the breed, even though it is known to be a carrier of hereditary disease. Once a general decision to apply genetical methods of disease prevention has been adopted, there is inevitably some discouragement of effort to discover veterinary methods of prevention of that disease. If, however, a satisfactory method of prevention or cure is discovered, then all the efforts of the breeders to secure genetical elimination are rendered at least partially superfluous. The decision to apply genetical methods of disease elimination is therefore one which should only be taken in cases where the mode of inheritance is very clear, where the disease or defect is itself of a serious nature, and where there seems to be no likelihood of finding a simple and inexpensive method of veterinary prevention or cure. Serious abnormalities should not be tolerated in breeding stock, but where these exist, genetical elimination should be planned in collaboration with the veterinary profession, as proper diagnosis and a knowledge of scientific breeding methods are essential. Selection against trivial conditions or those whose inheritance is uncertain (e.g. cryptorchidism), especially when conducted by breeders not really capable of correct veterinary diagnosis, is merely a waste of effort and liable to harm the breed by the elimination of good animals which need not have been sacrificed.

In concluding this chapter it may be useful to give the following list of the defects and diseases which have been discussed, together with the reported mode of inheritance of each. Those dealt with in other chapters are included in this list. The modes of inheritance quoted are those stated by the authors concerned, but most should be regarded rather as suggestions than as proven facts. As in so many other instances of genetical work on dogs, authors have been over-anxious to fit all their observations into a simple Mendelian scheme of inheritance, and have often failed to appreciate the inadequacy of their data, especially where only small numbers of animals were involved. There is, for example, no information of general validity on variation in susceptibility to any infectious disease, either between breeds or between strains within breeds, although such variation undoubtedly exists and most probably has a genetical basis in many instances.

TABLE II

LIST OF HERITABLE DISEASES AND DEFECTS

Condition	*Stated Mode of Inheritance*	*References (See p. 102)*
Abnormal larynx	Mendelian recessive (Skye Terriers)	Koch, 1935
Anterior pituitary dysfunction	Incompletely dominant to normal	Koch, 1935
Asthma	Not stated	Iljin, 1932
Calvé-Perthes disease	Not known	Moltzen-Nielson, 1937
Cataract	Mendelian dominant (Alsatians), also not stated	Hippel, 1930 Iljin, 1932
	Associated with retinal atrophy (Irish Setters)	Hodgman *et al.*, 1949
Coloboma	Not stated	Iljin, 1932 Kittel, 1931
Cryptorchidism	Irregular; common in dwarf and short-headed breeds	Härtl, 1938 Kittel, 1931 Koch, 1938 Whitney, 1948
Cysts of eyes	Not known (St. Bernards, also Newfoundlands)	Kittel, 1931
Deafness	Various, see text	Dechambre, 1932 Gray, 1933 Mitchell, 1935 Pearson and Usher, 1929
Distemper susceptibility	Not known definitely, but partially recessive in Bloodhound × Basset Hound and Bloodhound × Bull Terrier	Smith, 1939 Whitney, 1948
Eczema proneness	(*a*) Connected with coat type and breed	Witzigmann and Käb, 1937
	(*b*) Periodic, dominant in Pointers	Marchlewski, 1930
Epilepsy	Not stated	Iljin, 1932
Fissures of eyelids	Not known (St. Bernards, Newfoundlands)	Kittel, 1931
'Fits', proneness to	Uncertain	Whitney, 1948
Glaucoma	Irregular	Iljin, 1932 Whitney, 1948

Condition	*Stated Mode of Inheritance*	*References*
Haemophilia	Sex-linked recessive	Brinkhous and Graham, 1950 Hutt *et al.*, 1948 Merkens, 1938
Haemorrhage and periodic diarrhoea	Irregular, allergy involved (English Setters)	Ollerhead, 1939
Hairlessness	Dominant, probably lethal in homozygotes	*Journal of Heredity*, 1917 Kohn, 1911 Lëtard, 1930 Prinzhorn, 1921
Hare-lip	Doubtful whether inherited or not	Steiniger, 1940-42
Hernia, umbilical	Multiple recessive genes	Phillips and Felton, 1939
Hydrocephalus	Several recessive genes; Bulldogs and their descendants in crosses	Stockard, 1941
Kidney stones, predisposition to	Recessive (Dalmatians)	Trimble and Keeler, 1938
Microphthalmia	Occurs in homozygous 'merles' and 'harlequins' due to gene M (see text)	Mitchell, 1935 Whitney, 1948 Wriedt, 1925
Oestrual weakness	Partially dominant	Koch, 1935
Overshot jaw	Mendelian recessive (Dachshunds)	Grüneberg and Lea, 1940
	Recessive with modifiers (Cocker Spaniels)	Phillips, 1945
	Due to interbreeding short-faced types and normal breeds	Stockard, 1941
Paralysis	(*a*) In St. Bernard-Great Dane crosses	Stockard, 1936, 1941
	(*b*) Common in Toy Spaniels and Bull breeds	Gray, 1930
Pituitary cysts	Short-headed breeds (Bulldog types), recessive in crosses with other types	Obouissier, 1942 Stockard, 1941
Retinal atrophy	Mendelian recessive gene	Hodgman *et al.*, 1949
Rickets, proneness to	Irregular	Raghavachari, 1943 Stockard, 1941
Shyness, extreme	Dominant or partially dominant	Stockard, 1941 Thorne, 1944
Skeletal abnormalities	(*a*) Irregular, in descendants of affected French Bulldog	Klatt, 1939
	(*b*) Result of disharmonious breed crosses	Stockard, 1941
Spinal inflammations	Toy Spaniel and Bull types especially susceptible	Gray, 1930

Condition	*Stated Mode of Inheritance*	*References*
Tail, absent or short	Irregular, see text; in some instances dominant and lethal in homozygote	Briggs and Kalis 1942 Gates, 1909 Gossweiler, 1930 Lienhart, 1932 Rcheulishvili, 1938 Steiniger, 1940 Stockard, 1941 Uray, 1937 Vilmorin, 1913 Whitney, 1948
Tetany	Irregular, possibly recessive gene (Scottish Terriers, also occasionally in other breeds)	Klarenbeek *et al*, 1942 Thorne, 1944
Tooth number, reduced	(*a*) Incompletely dominant, associated with hairlessness	Gaspar, 1930
	(*b*) Recessive, several genes involved	Humphrey and Warner, 1934
Trembling	Irregular (Airedales)	Kollarits, 1924
Undershot jaw	Due to interbreeding short-faced and normal breeds	Stockard, 1941
Whelping troubles	Certain types especially common in certain breeds	Freak, 1948

V

BEHAVIOUR AND MENTAL ATTRIBUTES

UNDOUBTEDLY the very special position which the dog, amongst all domestic animals, holds in relation to mankind developed because of the behavioural and mental attributes of the canine species. If many dogs are now valued more for their appearance than their behaviour, this is a relatively recent and perhaps a passing phenomenon; the firm basis of the dog's place in civilisation, his privileged position as 'man's best friend' is dependent on his behaviour rather than on any physical characteristic, except perhaps his companionable range of body size. Yet, among nearly two hundred scientific studies of inheritance in dogs only a score deal specifically with mental attributes. Of these, the most comprehensive are those contributed by Stockard and his collaborators (1941), by Whitney (1929, 1932, 1948) and by Humphrey and Warner (1934). In a recent report (Scott and Fuller, 1951) on the first five years of a large-scale research project concerned mainly with behaviour patterns in dogs, it is stated that most of the genetic differences, except those relating to reproduction, are present by 10-12 weeks of age. As detailed evidence is not presented in the report, publication of the full results will be awaited with interest.

Stockard *et al.* (1941), in the course of observations on their numerous breed crosses, make reference in various instances to the general behaviour of the dogs. Thus they note the 'slow stupid' behaviour of many Bulldog-type individuals: this is associated with internal hydrocephalus ('water-on-the-brain'); the F_1 Griffon Bruxellois × Dachshund is described as 'nervous and restless like the Griffon, shy and snappy like the Dachshund'. Bulldog-Alsatians were 'vicious towards other dogs and extremely shy and wild while whelping'. In an attempt to make a more accurate assessment of the behaviour of the various breeds and crosses, the animals were tested by the conditioned reflex method.

This method makes possible very accurate testing of the response of each dog to the same carefully controlled experimental situation, but has only indirect significance for practical dog trainers, since the experimental situations with which the dog has to deal are very artificial and outside its normal life. For readers unfamiliar with the conditioned reflex method, a brief description may be of interest. The dog is placed standing on an experimental table where it can move about but cannot get away, and various 'stimuli' are introduced, such as the ticking of a metronome, the sound of a bell, rubbing the coat, and so on. These are associated with the arrival of some food, or with unpleasant results such as a slight electric shock to one foot, and in due course the dog responds to the 'conditioned stimulus' either by salivating, or by lifting the foot as the case may be. The time taken in learning, the effect of conflicting stimuli, etc., are recorded.

It is not possible or necessary to repeat here the detailed results of these tests, but in general they confirmed the less exact observations on the different behavioural types. Two pure types with opposite behaviour classifications were identified, the *lethargic*, of which the Basset was the typical example, and the *active*, typified by the Alsatian and the Saluki. The Alsatian × Basset F_1 were intermediate in behaviour and better balanced than either parent. In the F_2 there was a range of behaviour: those most like the Basset in form were most lethargic, those most like the Alsatian were most active, but among the rest were some more like the Alsatian in shape yet with behaviour more like the Basset. The Bulldog nature appeared to be a mixture of two extremes. It reacted with extreme violence to signals (whether they indicated pleasant or painful results) but became deeply inhibited and unresponsive during intervals between signals. The heightened excitability was shown by all F_2s with abnormal head and screw tail; some were untrainable and they turned out to have internal hydrocephalus. Extreme passive defence reactions were shown by three descendants of the Basset × Alsatian, and these animals were found to be quite untrainable. The behaviour of the Dachshund was similar to the Basset in these tests.

The conclusion was that the general type of behaviour is as characteristic of each breed as is its appearance, but that in cross-breds various blends, including extreme types of behaviour, occur.

In some cases, notably in Bulldog-like animals, there seemed to be a correlation between appearance (type) and behaviour, but in others type may resemble one parent and behaviour the other.

Whitney's approach to the problem was a different one, more directly connected with practical training of dogs. He analysed the behaviour of certain pure breeds into various component 'aptitudes' which, encouraged by training, are useful for hunting or other kinds of work, selecting for study those aptitudes which he considered least affected by training. As a result of these studies he lists the following pairs of alternative aptitudes and their genetic relation:

Trail Barking	Dominant to	Mute Trailing
Choppy Bark	Dominant to	Hound drawl
Hunting with head high	Partially dominant to	Hunting with head low
Water-going	Dominant to	Lack of it
Bird interest (interest in flying things)	Dominant to	Lack of it
Straight Hunting	Incompletely dominant to	Quartering
Smiling	Dominant to	Not smiling

Whitney, however, points out that although the dominance or partial dominance is shown in the first cross, further generations do not indicate any clear or simple Mendelian segregation of the mental aptitude, except possibly in the case of the 'smiling' tendency. Like Stockard *et al.* (1941) he found that in certain crosses F_2 puppies might resemble one pure breed in appearance but show behaviour characteristic of the other parental breed.

Marchlewski (1930), in crosses of German and English Pointers, found that the silent hunting of the English Pointer behaved as dominant to the yelping of the German Pointer. This contrasts with the dominance of the trail barking in Whitney's crosses of English Pointers with hounds. Marchlewski agrees with Whitney that hunting with head high is dominant to hunting with head low. Marchlewski found the placid disposition of German Pointers dominant to the more excitable nature of the English breed.

Other observers, besides Stockard *et al.*, have observed the occurrence of excessively shy and nervous dogs in experimental crosses. Krušinskiĭ (1938) obtained such animals by combining, through crosses, the shyness of wolves with the greater excitability of domestic dogs, and also by crossing the Alsatian with a Russian breed called the Giliatski Laika. Iljin (1941) also mentions the occurrence of extreme passive defence reaction in some of his wolf × dog crosses. Thorne (1944) found that 73% of the descendants (F_1 and F_2) of an extremely shy, fear-biting Basset Hound bitch were shy unfriendly animals, although the sires used were normal friendly dogs of other breeds (Saluki, Dachshund, Bulldog and Alsatian). Their behaviour was not modifiable by training, and Thorne suggests that it may have been due to inheritance of a single dominant Mendelian factor. Such abnormally shy individuals also occur occasionally in purebred stock, and it is the general experience of breeders that excessive shyness is likely to be passed on to the progeny. This danger is particularly great in breeding from an excessively shy or nervous bitch, since in addition to direct inheritance the behaviour of the mother is likely to teach the pups to react as violently as herself to the presence of strangers or other disturbing influences.

A matter of great interest to the gundog breeders is the possible inheritance of gun-shyness. This trait is widely believed to be incurable and strongly inherited; but many leading gundog trainers distinguish between 'gun-shyness' and 'gun-nervousness', the latter referring to dogs which show nervousness when first hearing shots, but which lose their fear when they become accustomed to the gun. (Dr Kelley points out that 'whip-shyness', i.e. fear of the cracking of a whip, is comparable to gun-shyness, and is inherited in some strains of Australian sheepdogs; some dogs, at first whip-shy, do become accustomed to the sound, whilst others do not.) No scientific studies on this matter in gundogs are available, but the work of Humphrey and Warner (1934) is relevant to the question. They found that each of their dogs could be assigned to one of three grades in regard to auditory sensitivity, and suggest that under-sensitivity may be due to a partially dominant gene 'N' whose allele 'n' when homozygous causes over-sensitivity (gun-shyness) as under:

Auditory under-sensitivity	genotype NN
Auditory sensitivity medium (normal)	genotype Nn
Auditory over-sensitivity	genotype nn

However, the three grades were not sharply distinct from each

PLATE VII (*a*)

PLATE VII. Instinctive Working Behaviour. (*a*) Border Collie 'showing eye' on sheep. (*b*) Pointer 'pointing' game.

(*b*)

other, and the authors suggest that this genetical hypothesis may be too simple. The same applied to tactual over-sensitivity (stick-shyness) which is independent of gun-shyness and may depend on a gene pair SS (under-sensitive), Ss (medium) and ss (over-sensitive). Both gun-shyness and stick-shyness were associated with sex, more males being under-sensitive and more females over-sensitive than would be expected if there was no effect of sex on the trait; this agrees with the general experience that bitches in all breeds tend to be more sensitive than dogs. Over-sensitive animals of both types were eliminated from the training and breeding programmes, and therefore four types remained:

(*a*) under-sensitive to both sound and touch
(*b*) under-sensitive to touch, medium to sound
(*c*) under-sensitive to sound, medium to touch
(*d*) medium to both sound and touch

Type (*c*) had the lowest percentage of low success scores as workers, and type (*d*) had the highest percentage of exceptionally good workers, but also many with low success, as this type is easily spoilt by unskilful training. Many dogs of types (*a*) and (*b*) had low success scores. Thus it was a worse fault for a working dog to be under-sensitive to touch than to sound.

Other behaviour traits, the inheritance of which was studied by Humphrey and Warner, included intelligence (ease of learning) which showed a negligible correlation between parents and offspring, as did aggressiveness; nevertheless, the latter quality decreased in the strain, apparently as the result of selection against over-aggressiveness. It was found that many degrees of aggressiveness occurred, and that both extremes were undesirable in all types of training, a moderate amount of aggressiveness being desirable; without it, the dogs lacked self-confidence and 'push'. Reduced initiative appeared to be correlated with the reduced aggressiveness of the later members of the strain.

The quality named 'distrust' showed increased incidence in one family, but was eliminated by selection from others, and is stated to be probably dependent on multiple factors. Distrust was found to be quite independent of shyness, such dogs showing no fear, but simply avoiding strangers and refusing to 'make friends'. Contrary to the demands of the 'Standard of Points' for the Alsatian, distrust was found not to be a desirable quality in police dogs, its only value being in army liaison work. The authors emphasise the importance of a reliable and friendly disposition in police dogs, so that they will not attack or bite unnecessarily.

Willingness in general, and the two special types, trailing willingness and unwillingness to retrieve, were all evidently inherited and 'ran in families', but inheritance was not of the simple Mendelian type; although possibly unwillingness to retrieve may have been due to a dominant gene.

Kelley (1951) reports that in his Border Collies ease of learning is at least sufficiently inherited to make selective breeding for it effective. He states that it is closely associated with tractability, which suggests that he is only concerned with the ease of learning to do what is required. Kelley's results thus do not necessarily conflict with those of Humphrey and Warner, since they also found that 'willingness' was strongly inherited, but they scored a dog's intelligence equally high whether it quickly learned to do as the trainer desired, or to avoid doing what was wanted.

Krušinskiĭ, in a series of studies (1938, 1944, 1946 *a*, *b*, *c*), found that another important gundog trait, the inborn tendency to carry objects in the mouth, was definitely inherited. Dogs with this tendency are of course more easily trained to retrieve than are dogs which lack it. He also studied the variation in the expression of 'passive defence' (cowering, running away, etc.), and 'active defence' (attacking, etc.), reactions as between different breeds, dogs and bitches, and the effect of rearing in isolation, and of drugs such as caffeine. His results are somewhat complicated, but indicate that dogs tend to stand up to nervous strain better than bitches, or, as he says, have 'stronger nervous systems'. Females show more marked passive defence reaction, but males show the strongest active defence. Krušinskiĭ makes the important suggestion that the mental adjustment or adaptation which occurs when fear is overcome (e.g. by the animal becoming accustomed to its surroundings) may eventually become inherited. Bleuler (1934) also states that training can lead to the inheritance of acquired instincts, but advances no evidence. The belief is, however, too widely held by experienced breeders and trainers to be dismissed, despite the lack of rigid proof complained of by geneticists. (See Chapter IX.)

Trimble and Keeler (1939) observed that when Dalmatians are trained to follow a carriage, some choose to run under the carriage with their nose close to the horse's heels, others run behind the carriage, whilst still others occupy intermediate positions. Each dog always runs in its preferred place, and this 'position preference' appears to 'run in families', i.e. it is inherited, but not in a simple Mendelian manner.

Croxton Smith (1941) reports briefly on the suitability of different breeds for Army work in Germany, as judged by the percentage successfully trained in each breed; Airedales, Boxers and Dobermann Pinschers were all 32% successful, whilst Alsatians were 10% lower. In Britain, the great majority of acceptable dogs were Alsatians, but Border Collies were the most suitable for carrying messages.

No exact scientific study appears to have been made of the inheritance of the very specialised and valuable behaviour-complex characteristic of the 'Trial-bred' Border Collie. It is clear that this behaviour can be inherited, and Kelley's work (see Chapter VIII) demonstrated that linebreeding can fix outstanding working qualities in a strain.

The behaviour pattern of Trial-bred Collies can be analysed into a number of components which may be found to be separately inherited. The main components are 'eye', 'power', and 'style' (which may be 'clapping', i.e. frequently lying down when checking, or 'upstanding').

The behaviour known as 'showing eye' seems to consist of two parts, the tendency to watch eagerly and to follow any moving object, and the tendency to check at a certain distance from the object of interest. The first tendency is obviously strongly developed in many breeds and individuals which show no eye (e.g. Greyhounds). It seems quite probable that the second tendency is the same as that which makes Pointers and Setters 'set' their game and it is notable that in working strains of Setters both 'clapping' and 'upstanding' styles occur. The connexion between eye and style is far from clear, but commonly Collies with an extreme clapping style tend to show excessive eye; dogs with excessive eye also tend to lack power, and powerful dogs with upstanding style do not show an extreme amount of eye. The position is extremely complicated because all grades of intermediate behaviour types can occur, only rare individuals being clear cases of 'over-eye', 'no eye', 'extreme clapper', etc.; furthermore, behaviour can be greatly modified by training, e.g. eye can be developed by teaching the dog to work at a distance from the sheep. Kelley (1947), using a scoring system 0 = 'no eye' to 6 = 'over-eye', found that the mean score of the progeny was close to the mean of the parents' scores, and suggests that the inheritance of 'eye' is controlled by 'many additive genes'. In the examples he quotes both parents scored fairly high, and the score of each pup was identical with that of one or other of its parents. In a few matings observed, crossing Collies showing eye with those lacking it resulted in the progeny all showing some degree of eye. (Unpublished data—M.B.) Extremely upstanding style seems seldom to be passed on to progeny out of a 'clapping' mate, but the degree of clapping may be considerably lessened, i.e. the offspring show an intermediate range of behaviour; frequently some 'clap' as much as the 'clapping' parent, but it is less likely that any will have a fully upstanding style. Eye and clapping might thus be described as partially dominant in inheritance, if these observations are correct. However, no exact data are available; elucidation of the question would require that whole litters be reared and trained by one person in order to eliminate differences of behaviour due to training.

There is also the question of whether special 'interest' in certain things is inherited. Whitney, in discussing the inheritance of what he calls 'bird interest', describes the dogs as being interested in birds, butterflies and any flying object. It might thus be a general interest in small moving objects, coupled with the tendency to hunt with head high, causing the dogs to notice flying creatures. Bird dogs, unless checked by training, will also pursue ground game; young Border Collies watch birds and butterflies and show eye on anything that moves, even ripples on a pool. Yet, despite this generalising of behaviour, there does seem to be some special attraction to particular animals. Thus, untrained Spaniels become much more excited by their first scent of a rabbit than do Pointers or Collies, though all show some interest. Pointers are more excited by bird-scents, and are also more willing to carry feather than fur. A good Collie can be trained to work anything before it has seen sheep, but many are reluctant to work cattle once they have worked sheep; young pups have been observed to smell about where sheep have been, picking up bits of wool and sheep dung, whilst gundog puppies showed no interest. Dalmatians are reputed to have an inborn love of horses.

VI

COAT, SKIN AND COLOUR

THE great range of coat type in dogs, and the existence of many gradations intermediate between the extreme types, suggests at once that inheritance is likely to be of a multifactorial rather than a simple Mendelian kind. This is in fact found to be true, although a fairly simple relationship does exist in certain cases. Thus, in general, short coat is dominant (or epistatic) to long, straight or wavy types, and recessive or partially recessive to 'wire coat' types. American workers (Scott and Fuller, 1951) report that good evidence has been obtained for the existence of at least 13 genes influencing coat colour and texture.

The characteristics of each coat type can be analysed into a number of components most of which appear to be inherited independently of each other. Those components include length, coarseness, texture, wave or curl, density (i.e. number of hairs per unit area of skin), medullation (the presence or absence and type of the central hollow core in the hair). In crosses between different breeds segregation and recombination may cause the appearance in the F_2 and later generations of coat types not found in either parent. Many examples of this will be found in the work of Stockard *et al.* (1941). Whitney (1948) found that in the F_1 density tends towards the least densely-coated parent, straight hair is partially dominant to curly or wavy, and coarseness dominant to very fine coat texture. The presence of tail feathering was dominant to its absence in crosses between Irish Water Spaniels and Irish Setters or Cocker Spaniels.

In crosses between wire-coated and short-haired breeds the F_1 frequently shows the 'beard' or bristly hairs on the muzzle and chin, which are conspicuous in most wire-coated breeds; the coat is short elsewhere and straight, but with the harsh texture of the wire-haired parent. In the Griffon Bruxellois, however, Little (1934) found that short, smooth coat behaved as a simple recessive to wire coat.

Pullig (1950) reports the occurrence of unusual hair whorls in a strain of American Cocker Spaniels. The whorls were in most instances present either on both shoulders or on both flanks, but in a few dogs only one whorl was present, or the location was on the head or neck. Study of the pedigrees indicated that the condition was inherited, and probably due to a recessive factor which was not sex-linked. There was a possibility of linkage between the gene causing whorls and that causing red coat colour but the data were insufficient to confirm this.

Whitney (1948) discusses a fact which must be evident to any breeder of particoloured dogs, namely that pigmented hairs on such animals frequently grow much longer than neighbouring white hairs, and that black hairs grow longer than yellow pigmented hairs. Chocolate (liver) hairs grow as long or nearly as long as black but tend to have a softer, more 'woolly' texture. Yellow or red hairs tend to be finer and more silky than black ones. Such comparisons can, of course, only be validly made between closely related dogs of the same breed.

There are also breed differences in the frequency and mode of shedding the coat. In some types, especially short-coated breeds lacking undercoat, moulting is a prolonged process and indeed a few hairs are shed all the year round. Such coats do not seem to be very secure at any time, in that vigorous brushing or plucking will release many hairs even when the dog is not moulting. Double-coated breeds on the other hand have two defined moulting periods during the year, and in these breeds there are therefore some months in which the dogs are so 'out of coat' as to be unfit for show. This applies mainly to the autumn moult in which the long hairs of the outer coat are shed. In the spring it is mainly the undercoat which is lost and the effect on the dog's appearance is less. These differences in shedding seem to be attributes of the different coat types, but no exact information on the inheritance of moulting behaviour is available.

There is a very widespread belief that dogs do not sweat from the general body surface but only from the pads of the feet and 'the tongue' (i.e. evaporation from the mouth when panting). Nevertheless, Jäkel (1940) found plentiful sweat-glands, which appeared to be functional, in all parts of the skin of the eleven breeds examined, and it seems probable that the popular belief is, at least in part, a fallacy. In Bulldogs and Poodles the sweat glands were found to be particularly large.

Hare (1932) describes a congenital abnormality of the skin of Rhodesian Ridgeback dogs which was probably inherited since an affected dog mated to his normal sister sired four normal and two affected pups. The abnormality consisted of a narrow and deep invagination of the skin in the mid-dorsal line on the neck, withers or croup.

Several workers have studied the inheritance of hairlessness in crosses between 'hairless' breeds and normal dogs (*see* Table 2). The hairless condition usually behaves as a simple Mendelian dominant, but in one instance where hairlessness occurred within a normal breed (Poodles) no definite evidence concerning inheritance of the condition was obtained (De Zulueta, 1949).

PLATE VIII. Yellow × Sable gives Black Puppies. A golden sable Collie dog, the same colour as the Show Collie bitch (*a*) was mated to the black and white Border Collie bitch (*b*) whose dam was black and tan with white markings. Their offspring comprised three black and white, two black and tan with white, and one shaded sable bitch. The latter was mated to the pedigree Yellow Labrador shown with her and their five black and white puppies (*c*), of which none had any tan markings. This result was predicted on the basis of the 'Extension and Agouti Series' theory of canine colour genetics (see Chapter VI). The gene formulae are as follows:

a^ya^tEE × Aa^tEE
(sable dog) (black bitch)
Aa^yEE Aa^tEE a^ya^tEE a^ta^tEE
(black) (black) (sable) (bicolour, i.e. black-and-tan)

a^ya^tEE × $AAee$
(sable) (Yellow Labrador)
Aa^yEe and Aa^tEe
(all black)

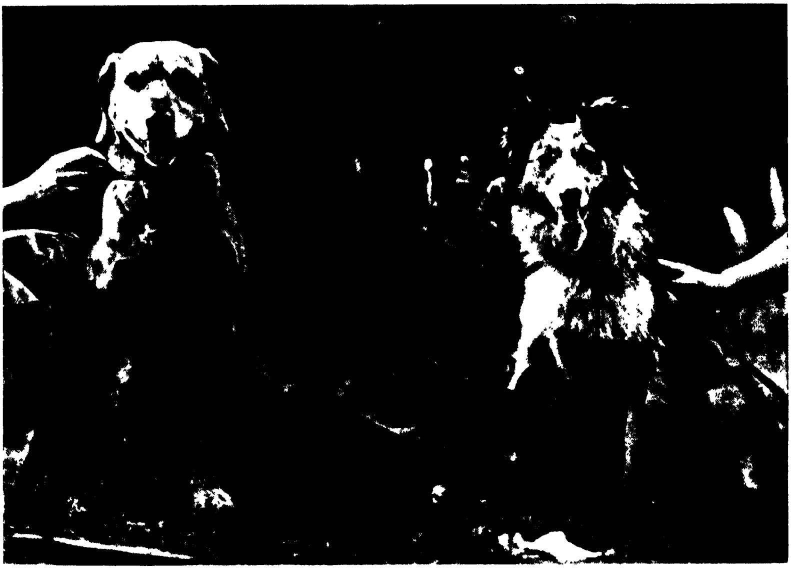
The white markings are unimportant in this experiment as it is the inheritance of black, black-and-tan (bicolour), sable and yellow that is being considered.

PLATE VIII

(*a*)

(*b*)

(*c*)



It is a curious fact that, although more scientific papers have been published on the subject of coat colour in dogs than on any other aspect of canine genetics, and despite the apparent ease of observing and recording coat colour, yet the whole subject remains full of uncertainties and contradictions. Each additional contribution only seems to make confusion worse confounded. In a book intended to be intelligible to the practical dog breeder it would be out of place to attempt to review every contribution or to join in detailed controversy, although it is essential to indicate which facts are generally agreed upon by scientists and which are still the centre of debate.

It can be said, from work on other mammals, that the pigment is composed of chemical substances called melanins, and is usually deposited in the form of granules in the skin and hair. This process begins some weeks before the pup is born, but continues after birth. In albino animals it is believed that some chemical substance necessary to the formation of pigment is lacking, or, in the case of partial albinos, present in very limited quantity. Under a microscope the pigment granules of 'black' hair appear very dark brown or black, whilst those of red or yellow (fawn) hair are smaller and appear yellow. In dogs two kinds of black pigment have been observed by the author: the commonest kind is deposited mostly in fairly large granules, although there is a wide range of granule size from small to large; in the second type all the granules are small and even in size. After bleaching with an oxidising agent such as hydrogen peroxide, the pigment granules of black dogs appear the same as those of yellow dogs. This suggests that 'yellow' pigment may be formed from black pigment by a further chemical process. This is supported by the fact that yellow dogs usually have black pigment in their skin (including the eyes and nose) and that the amount of tan on a black-and-tan dog continues to increase after birth. When a tan dog does not have black pigment in the skin, the skin pigment is not yellow but chocolate. Under the microscope chocolate (liver-coloured) hairs are seen to contain light brownish pigment in smaller granules than those of black hairs, showing a special clustering together, and very regular in size. It appears that the chocolate pigment can be oxidised to yellow just as the black pigment can, hence we get black-nosed tan and liver-nosed tan dogs in several breeds. This bleaching to yellow takes place only in the hair, never in the skin (unless possibly in the eyes but this is doubtful). However, not all brown-nosed tan or yellow dogs have chocolate skin pigment; the 'yellowing process' appears also to reduce the quantity of pigment in both skin and hair, so that often the nose becomes a rusty-brown colour, which is quite distinct from the true 'liver' nose to an experienced observer. The blackness of a yellow dog's nose often varies at different periods in the dog's life (we all know that perverse black nose that always goes brown on the day of a show!); it is probably sometimes due to partial bleaching by sunlight, in the same way that some black or 'blue' dogs tend to go 'rusty-coated' in the summer.

The pigment is deposited in the hair as the hair grows, and not afterwards; therefore, if the pigment-forming process in the hair-root changes when a hair is partly grown, that hair will come out banded, partly one colour and partly another. The best known type of banding is that found in the coat of wild rabbits and many other wild animals including wolves; scientists usually call this type 'agouti pattern' after a wild rodent called an agouti. It is seen in many Alsatians, Elkhounds, etc., and is a very complicated pattern, varying on each hair and shading from darker on the back to lighter underneath; the majority of the hairs have black tips, then a yellow or whitish band, below which there may be another dark band although often the colour gets continuously paler towards the skin. Agouti-coloured puppies are born almost black, as only the tips of their hairs show at birth.

One gene about which all workers seem to agree is the one which makes the dog produce chocolate-coloured pigment instead of black. This is recessive to its allelomorph which when present allows black pigment to be formed. These genes were named B (black) and b (chocolate) by Little in 1914 and their relationship seems to be definite and simple, the gene b always behaving as a simple Mendelian recessive to B in every experiment that has been reported, in numerous breeds and their crosses. A dog which has the formula bb apparently cannot produce any black pigment anywhere in its body; it is therefore no use breeders demanding a black nose or very dark eyes in such dogs, and in fact in all breeds in which liver coat colour is permitted, the 'Standard of Points' permits brown noses and lighter eyes with this coat colour.

Rather illogically, the same concession is not always made to liver-nosed yellow dogs, e.g. in Cocker Spaniels.

There are only two other coat-colour genes about which there seems to be no debate, one the 'merle' gene, which will be dealt with later, and the other the gene for 'blue dilution'. The latter gene was also named by Little (1919) and he gave it the symbol d as it is also a simple recessive to its allele D. In a dog which lacks D, i.e. has the genetic formula dd, the pigment granules are clumped into very distinctive aglomerations, larger and much more definite than those produced by the gene b. This clumping certainly affects black and chocolate pigment and some tan (yellow) pigment, but there may be a kind of yellow that is not affected. The effect on black pigment gives the familiar 'blue' of Greyhounds and Great Danes, the 'silver' of Poodles, etc. With chocolate pigment, it produces the rather pinkish shades known as 'liver' in Bedlington Terriers and 'apricot' in Poodles; with yellow pigment to work on, it produces blue-fawns. Blue-dilution affects the pigment in the skin, eyes, etc., as well as in the hair. This is the reason why 'blue' dogs have slaty noses and smoky eyes. This is the available information about blue-dilution; what is not known is the cause of the many shades of blue. Probably the shade of blue of dd dogs depends on the gene complex as a whole, as it does in mice and other rodents, but possibly D is not always fully dominant to d, so that the very dark 'blues' may be heterozygous Dd.

The tan, yellow, red, fawn and golden colours of dogs are the ones that have set scientists the greatest puzzle. There are so many shades, and, to make matters worse, dogs may be partly tan and partly black or liver. (No dog has ever been recorded as partly black and partly liver, or partly black and partly 'blue-dilute'.) In different breeds the same shade has different names; indeed, this may be true even within a breed: a solid-coloured Cocker may be called red, or if lighter, golden, but if it is a particolour the corresponding shades will be called orange-and-white or lemon-and-white. In addition the term 'brown' is sometimes used for yellowish dogs and sometimes for liver-coloured ones; and very few breeders seem able to recognise chocolate-sables (see below).

The oldest theory of the inheritance of tan colours has been built up in the course of many years' work by numerous scientists among whom Little, Sewell Wright and Iljin made especially important contributions. According to this theory, there are two independent allelic series both of which include tan-producing genes. The first, or agouti series, contains several alleles. Combining the suggestions of various authors, and thus slightly modifying Iljin's original list, these alleles include the following:

A	dominant black
a^g	agouti (wolf grey)
a^y	sable or 'dominant yellow'
a^s	saddle (black or liver saddle with extensive tan markings on legs and head)
a^t	bicolour (black or liver with restricted tan markings on feet and muzzle)

There may also be a dominant bicolour A^t in Dobermann Pinschers (Iljin 1931).

Apart from the dominant black, all these coat colours tend to be lighter on the extremities and under-side of the dog than on the back; they are all darker at birth than when adult. Although dominance is in the order as listed, it is not complete (except the black); thus a Collie with the formula $a^y a^y$ is a 'clear golden sable' (Whitney questions this), but one which carries bicolour $a^y a^t$ has a large amount of black hair scattered through the 'golden' on its back and head. In long-haired dogs, such as Pekingese, dark sables are often called 'brindled'. Throughout this series liver-colour may be substituted for black, giving liver-and-tan or chocolate-sables, etc. The latter often appear red-sable with liver noses, but close examination reveals the chocolate colour in the hairs on the back.

The second allelic series was named the extension series by Little (1914) and may contain three or four alleles:

E	dominant black
E^b	brindle
e^m	fawn with black mask
e	fawn without black mask

There are many criticisms of this theory, but it does enable one to predict the results of colour crosses with reasonable accuracy, although exceptions occur. A few examples favourable to this theory will be given before discussing other theories.

(1) Many breeds are homozygous AA so that the agouti series

does not appear to affect them, and their 'yellow' colours are all of the ee type. Thus in Great Danes, Greyhounds and Poodles the blacks are all of the formula AAE– and the fawns are AAee. Black behaves as a simple dominant to fawn. Brindle, in Danes and Greyhounds, is recessive to black but dominant to fawn; fawn with a black mask is probably dominant to fawn without a mask. The genes e^b and e^m are absent from Poodles. In Cocker Spaniels the reds and goldens are ee, and the plain blacks EE, but in this breed two genes of the agouti series occur, namely A and a^t. A black Cocker is A–E– and a black-and-tan one is a^ta^tE– (Barrows and Phillips, 1915).

(2) Show Collies are homozygous EE and contain only two alleles of the agouti series, namely a^y and a^t. Sables are a^ya^y (golden sable) or a^ya^t (shaded sable); tricolours are a^ta^t.

If this theory is true a number of deductions can be made and tested by breeding results:

(1) Reds or fawns of the type ee when mated together should never throw blacks or brindles, but only reds or fawns. All breeders of the breeds concerned know that this is substantially true, although not without exceptions.

(2) Two 'bicolours' (black-and-tans, liver-and-tans, or blue-and-tans) mated together should throw all tan-marked pups. This is also proved by general experience in many breeds, but if any red or yellow pups occur the tan markings cannot as a rule be distinguished on them.

(3) If there are two genetically different types of 'yellow' dogs, the type with the formula A-ee and the type with the formula a^ya^yEE, these two mated together should throw nothing but black puppies of the formula Aa^yEe. The illustration, Plate VIII, shows a yellow Labrador dog (AAee), a dark sable Collie bitch (a^ya^tEE) and their five black puppies, which have white markings but no tan or yellow on them.

Where breeds or crosses involve several members of both the agouti and the extension series the matter becomes extremely complicated. It would appear that the genes ee when present produce complete yellowing of all black pigment in the coat, so that no matter what members of the agouti series are included in the formula an ee dog will be yellow to red in colour. The factor for brindling e^b appears able to affect all yellow areas with dark stripes, so that a 'saddle' or 'bicolour' dog may have brindled face or legs. However, in some instances, especially in Bull Terriers, brindle appears to be epistatic to bicolour and sable.

Whitney (1948) advances evidence that the factor for brindling may not be an allele of E and e, and suggests that it is an independent dominant gene whose effects can only be seen in yellow dogs. This would explain the 'ghost brindling' seen on so-called 'black-brindle' dogs.

Whitney also suggests a rather different theory of the relation between the various yellows and bicolour and saddle patterns. He proposes two alleles, T or t, both of which inhibit black, producing tan coats, and an independent dominant gene S which produces the bicolour or saddle pattern on dogs of formula tt but not on dogs carrying T. If both the genes T and t inhibit black it is not clear how any black dogs are supposed to occur, but Whitney of course knows of, and in other connexions frequently refers to, the dominance of black over almost all other colours. To clarify his theory it would be necessary either to introduce a dominant black as an allele of T and t, or an epistatic black whose allele would presumably be a third kind of tan, or possibly the saddle gene. This is an example of the common error of forgetting that every dog must have one or other allele of every gene on every locus, and of making statements without adequate proof. Whitney gives no clear evidence against the older theory which appears to explain all his examples, with one exception, as well as his own theory does. The exception is a litter of brindle pups produced by mating a brindle Dane to a bicolour Bloodhound. On the classical theory the offspring should have been black, $e^be^bAA \times EEa^ta^t$ giving Ee^bAa^t pups. On the other hand Whitney's theory fails to explain the occurrence of nothing but black (not black-and-tan) puppies from the mating of the yellow Labrador to the sable Collie mentioned above.

Another theory of coat colour inheritance has recently been advanced by Winge (1950), but it is extremely unsatisfactory and speculative, although it does work out quite well in the breeds in which he specialised. He gives no explanation of his complete overthrow of the older theory, and uses long-established symbols in entirely new meanings without any apology to the earlier users of those symbols. Thus he states that the gene for brindle is allelic to bicolour and recessive to it, and suggests a whole allelic series including these, without giving any proof that they are really alleles of each other. After all this, Winge's theory does not explain the totality of known facts any better than any previous theory, although he does offer a possible explanation of the occasional occurrence of black pups without tan markings from the mating of two bicolour parents. This could also be explained on the older theory if two genes for bicolour, one dominant and one recessive, are included in the agouti series (p. 56).

It is clear then that none of these theories can account for every result that has been reported from matings involving dogs with partly or wholly yellow-coloured coats. The answer is probably to be found in physiology rather than genetics, exceptional conditions of temperature, diet, or other circumstances being probable causes of unusual manifestations of the 'yellowing process'. In the great majority of matings, however, the results will be in accordance with the older theory of the agouti and extension series, as indicated in the next chapter.

A third series of alleles, the existence of which has been supported by several researches, is known as the albino series, with symbols as follows:

C = colour factor, without which little or no melanin can be made, whatever other genes of any other series may be present.
c^r = partial albinism, which in the heterozygous state (Cc^r) reduces red to chamois without affecting black pigment.
c^d = white coat with dark nose and eyes.
c^b = pale greyish ('cornaz') coat with pale blue eyes.
c = total albinism, pink nose and blue eyes.

The order of dominance is probably as given, but the relation of c^r to c^b is not certain. Probably dominance is not complete and the effect on yellow pigment is greater than on black pigment. Pigment in the coat is more strongly affected than pigment in the skin (including the eyes). Such colouring as is present in dogs lacking C tends to be strongest at the extremities (nose, feet and tail, also ears) taking the form of a yellowish or greyish tinge in the coat; however, where the animal has inherited white markings (see below), the slight pigmentation may be in patches on the head and body.

The earliest study of albinism in dogs, by Pearson and Usher

(1929), raises several interesting matters which have not been further investigated by later workers. They describe three types of albino (in Pekingese and Peke-Pom crosses) which entirely lacked melanin granules; albino white, or 'dondo'; 'lilac', described as pale millboard in colour with albinotic eyes; and 'cornaz', with diffuse pigment all over but not darker at the extremities. In addition there were 'skewbald cornaz' with white markings. Then there were whites with dark eyes and noses, which tended to develop a yellow tinge at maturity, and were very delicate. Curiously enough, the dondos, with no pigment, were the hardiest of these types. Matings between the various types gave results which suggest that more than one gene pair was involved. Thus although cornaz × cornaz gave only cornaz pups, dondo × dondo gave dondo, cornaz, and skewbald cornaz, as if dondo were dominant to cornaz. Yet pups with fully pigmented coats and eyes occurred in litters from cornaz × skewbald cornaz and cornaz × dondo, but never from dondo × dondo. It appears from this that cornaz and dondo were not due to allelic genes. The authors point out that in their records the first litter contained more variation in coat colour than did later litters from the same parents; they also suggest that fertilisation is selective and not random. In the light of this work the albino series of allelic genes, as given above, must not be regarded as certainly correct.

At this point it will be appropriate to discuss the so-called 'colour-paling' in Alsatians. It seems clear that several alleles of the albino series are present in the breed, including C (normal pigmentation) c^r and c^d. The white Alsatian with black nose and dark eyes will have the formula c^dc^d, its coat will be pure white, not cream, and if mated with coloured dogs which are homozygous CC, its descendants will either be pure white like itself or normally coloured. It is the other alleles of c^d which cause real 'colour-paling'. If the order of dominance in the albino series, as given, is correct, the white Alsatian may carry c^b recessively, but not c^d, and it is doubtful whether the c^b gene is present in the breed. Normally the pure white Alsatian will not contribute to 'colour paling', but the cream dog will. However not all colour paling is due to genes of the albino series. Iljin (1932, 1934) points out that the yellow bands on agouti-marked hairs may vary in colour from rich red to palest straw-colour, and this is true also of the tan markings on bicolour and saddle-marked dogs. He suggests three allelic genes: Int, int^m, and int, of which the dominant one, Int, produces the palest colour, while the homozygous recessive int int produces 'bright yellow'. 'Colour paling' can thus be partly due to the presence of the gene Int. Another gene, namely blue dilution dd is also found in 'colour paling' lines and produces paler coat colour.

In view of the numerous ways in which pale coat colours may be produced it appears absurd to assume that 'colour paling' is necessarily associated with constitutional weakness. There is some evidence that some genes of the albino series do reduce vigour, so that cream animals may be suspect. The pure whites seem to be perfectly normal constitutionally, and it is quite probable that the gene causing this (recessive white with black nose) is not really one of the albino series.

Of great interest is the dominant factor known by the symbol M which is associated with merle coat colour. Dogs heterozygous for this factor have the merle coat colour and often 'wall' eyes, but are otherwise normal; if two merles are mated together, however, a proportion of their offspring will be white or almost white in coat colour, with various abnormalities including deafness, blindness and structurally abnormal eyes. These are the homozygous MM individuals. Dogs without M, i.e. of the formula mm, are normal, and their colour is determined by the rest of their gene complex. The effect of the M gene in the heterozygous condition is to reduce the pigmentation in some areas whilst leaving flecks and patches intensely pigmented. If, apart from the M gene, the dog is genetically black-and-tan, the result is a blue-merle, as seen in, e.g. Collies, Dunkerhounds, Dachshunds. In sables it produces a dog with 'golden' patches on a cream background. Its effect on the Extension yellow, ee, has not been described and possibly it does not affect this type of yellow. No report of the appearance of merle hairs under the microscope has been made, so its exact effect on the pigment is not known. In Great Danes, the same gene seems to be responsible for the colour known as harlequin, although true merles also occur in the breed. Harlequins are usually bred by mating a harlequin to a black or a blue, and merles are said to occur when fawn or brindle blood is introduced into a harlequin strain. Possibly the gene M restricts black pigment more than yellow; it does not appear to affect the tan markings of bicolour dogs. Unfortunately Little (1919) who studied coat colour inheritance in this breed, ignored the existence of merles, presumably because breeders do not register them since the colour is not accepted in the show ring. Harlequin colour may be the result of interaction between the gene M and some other gene not yet identified. Breeders should note that if harlequins or merles are mated together or to each other, about one quarter of their offspring may be expected to be white with abnormalities of sight and hearing; such white pups should be destroyed at birth unless required for special purposes.

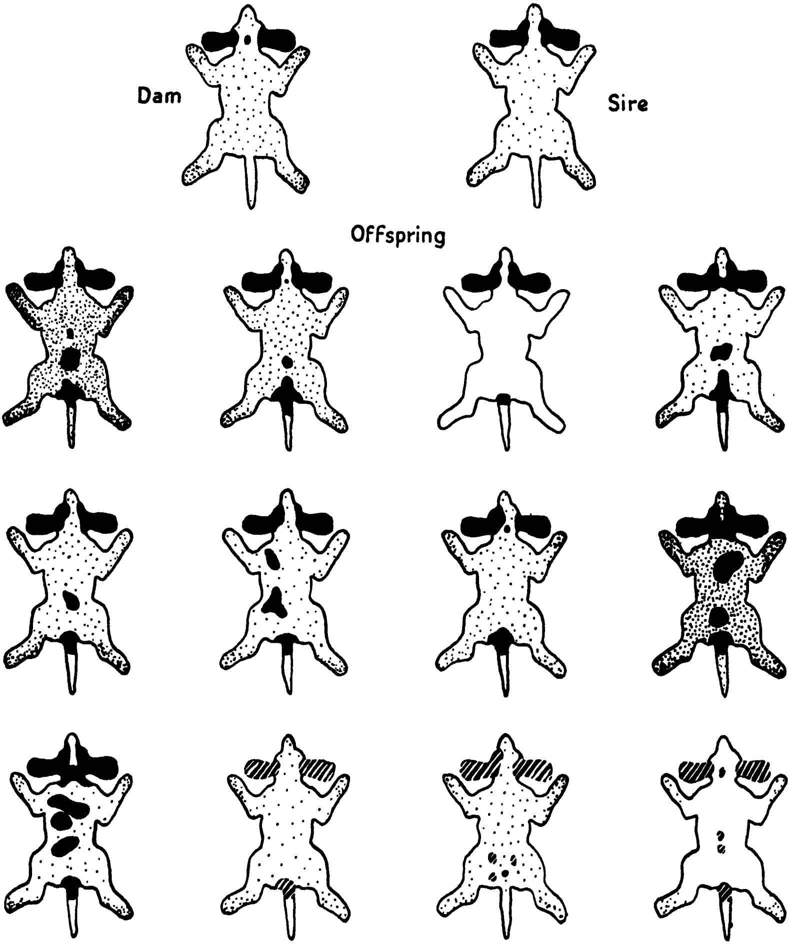
We have now completed our review of the genes reported to affect the shade of colour of the coat, and must turn to those affecting the distribution of coloured and white areas. Obviously, a white area is one in which for some reason no pigment granules of any colour have been formed. Scientists, working on mice, rats and rabbits, started the tradition of referring to white markings as 'white spotting' and using the symbol s. The kind of white markings to which this symbol is applied in dogs are recessive or partially recessive to solid (self) colour. A good example of a study of this kind of white is that of Warren (1927) on Greyhounds. These dogs are usually described when registered as either 'no white', 'coloured and white' or 'white and coloured'. Warren found that less white behaved as dominant to more white, i.e. dominance was in the order self, coloured and white, white and coloured. This has been attributed to three allelic genes, S, s′ and s, but this is not a very satisfactory hypothesis. We know that in breeds such as Racing Greyhounds, in which the amount of white is not considered important so that there is little selection for or against white by breeders, all degrees of white marking occur, from pure white to totally coloured animals. Allen (1914) points out that as the amount of white increases, the coloured areas seem to retreat before it in a rather orderly manner. If only a little white is present it is sure to be found on the chest, the toes, perhaps the belly, the muzzle and the tip of the tail. Any experienced breeder would be astonished to find a black puppy with a white spot in the middle of its back, or a white ear, and no other white markings. When more extensive white markings occur they spread up the legs, and round the neck, as often seen in Show

Collies and St. Bernards. With still more white, the coloured areas form a series of separate patches, the exact position of which varies, and yet there is a certain regularity. Thus the last part to lose pigmentation is the head, especially the ears, and another very persistent patch lies at the root of the tail. Allen called these areas 'centres of pigmentation' and believed that pigmentation of each 'centre' was inherited independently of the others. Marchlewski (1930) states that in Pointers a coloured spot on the occiput is inherited as a simple dominant to its absence, two parents without a 'head dot' never throwing puppies with the 'dot', and this seems to be true also in Cocker Spaniels (where there is much pigmentation of the head the ear-patches extend to meet the 'head dot' which cannot then be distinguished). No simple rules for inheritance of other 'pigmentation centres' can be given, but there is certainly a tendency for puppies not to have very much more extensive pigment patches than the most marked of their parents. Thus two lightly marked parents will not throw self-coloured or nearly self-coloured puppies, although some of their pups may have more colour than the parents had (Fig. 1). The colour with which the patches are pigmented also appears to affect the size of the patches, yellow pigment tending to reduce their size whilst they will be larger if the pigment is liver and larger still if it is black. It is for this reason that it is easier to breed a white Cocker Spaniel from liver-nosed lemon-and-white parents than from any other colour. Mrs. Barnes' well-known efforts to establish a strain of white Cockers were made more difficult by her insistence on black noses and dark eyes (Barnes and Barnes, 1946).

In Fig. 2 the most important centres of pigmentation in the dog are indicated and numbered approximately in order of persistence. Centres 2 and 3 are median in position, the others can all occur on both sides of the dog, although often one side patch is present and its opposite patch is missing. Centres 3 and 3a are about equally persistent, and the same is true of 4 and 4a, which are lost more readily than 3 or 3a.

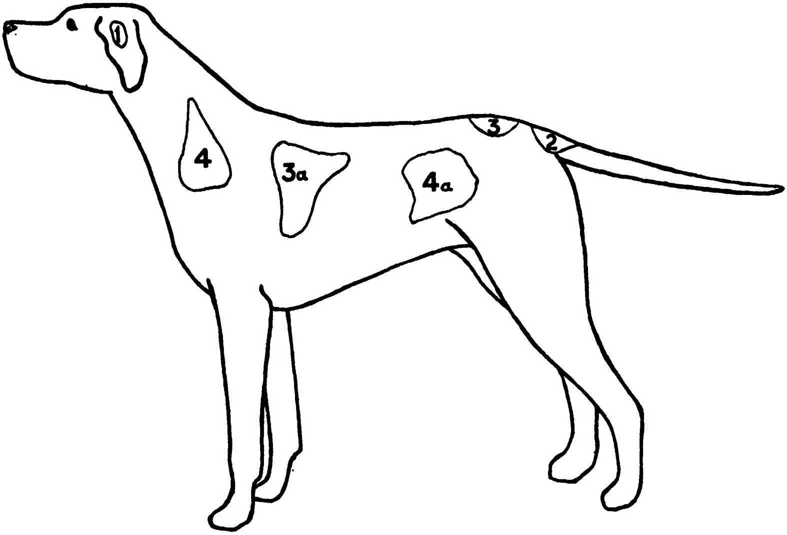
In breeds where a certain pattern of coloured and white areas is deemed desirable even many years of persistent selection have failed to establish absolute regularity of the pattern. Yet there is much less variation than in breeds in which no such selection has been practised. The puppies of perfectly marked parents range from those with an excess of white to those which have somewhat too little, but most of them are not far from the orthodox pattern for the breed.

FIG. I: Inheritance of Pigmented Patches and Roaning



In many dogs the white areas between coloured patches are not pure white, but contain small coloured spots (ticking) or coloured hairs intermingled with the white hairs (roaning). In some breeds such as Cocker Spaniels there seems to be a complete range from heavy roaning to light ticking, and the greater amount of colour in the white areas behaves as incompletely dominant to the lesser amount (Fig. 1). Thus, two Cockers with clear white markings will not throw roaned puppies, but a dark roan mated to a clear or lightly ticked animal will throw dark roans in the litter. The factor for roaning can be carried by a self-coloured dog but then its effects cannot be seen, unless there are small white markings on the feet and chest, in which case these will become roaned. It is characteristic of this type of factor that the puppies are born with pure white markings which become roaned or ticked after a few weeks or even months; also the roaning or ticking is always heaviest along the underside of the dog and on the feet and muzzle, and it is on these parts that it develops first. In this distribution it resembles the tan markings of bicolour dogs.

FIGURE II: Centres of Pigmentation



The spotting of Dalmatians is almost certainly one form of this kind of ticking.

Whitney (1948) states that a similar ticking is inherited recessively in English Pointers. Certainly, two Pointers with clear white markings can throw ticked pups—in one case a litter of six from such parents all became heavily ticked (author's personal observation). The ticking of the German Pointer (Short-Haired Bird Dog of Winge, 1950) is inherited as a dominant.

Whitney describes some of his experimental crossbred dogs which had clear white markings with patches of roaning as well as solid pigmented patches, and suggests that there may be two kinds of white markings, only one of which can be affected by the roaning factor.

Two other kinds of white have already been discussed, namely albino white and the white which results from the homozygous conditions of the 'merle' gene M. There is also the white which behaves as a simple recessive to coloured, giving a pure white coat with black nose and eyes, which is found in Poodles, white Alsatians, occasionally in Collies and Chows, and probably Samoyeds and West Highland White Terriers. As already discussed, it may or may not be one of the albino series of alleles (white dogs tinged with cream also occur in these breeds, probably due to another allele of the albino series). These white dogs are indistinguishable from white dogs resulting from the extreme maximum of white spotting, but they do not throw puppies with patches of colour as the latter type always do. White Bull Terriers are probably of the recessive white spotting type, since they often throw slightly 'marked' dogs.

Finally there is a type of white which behaves in inheritance as a simple Mendelian dominant, inhibiting the production of pigment in the coat, though the skin, nose and eyes are pigmented. This gene has been given the symbol W (Iljin 1932).

Eye Colour

In dogs carrying E–, B–, D and mm, eye colour is seen in three shades, dark, medium or hazel, and light yellow, and these may depend on three alleles with dominance in that order (Burns, 1943; Humphrey and Warner, 1934; Warren, 1927). As these refer to the colour of the iris, they may be called Ir, ir^m and ir^y. Dominance is probably incomplete, giving intermediate shades in heterozygotes. The inheritance of these shades is independent of coat colours, but several pigment factors, as already mentioned, also affect the eye colour; these are dd (smoky eyes), bb (rendering Ir eyes dark chestnut, ir^m eyes hazel, ir^y extremely pale yellow), cc and c^bc^b (producing albinotic blue eyes), and M (giving wall-eye if a patch of restricted pigment happens to cover the eye—an alternative hypothesis is that there is a separate factor which produces wall-eye and is closely linked with M). In addition Iljin (1926, 1928) reports a factor 'ruby eye' which affects the pigmentation of the retina in such a way that when a beam of light falls on the eye in darkness the light reflected is red instead of yellowish-green; it is recessive to normal condition. The condition of progressive retinal atrophy also alters the colour of light reflected from the retina in afflicted Red Setters, increasing the reflectivity of the tapetum and reducing the orange-yellow component so that the colour is more definitely green (Hodgman *et al.*, 1949).

Colour of Skin, Nose and Toe-nails

Pigmentation of the skin has already been mentioned in discussing the different effects of certain genes on the skin, nose and hair. Pigmented spots and patches may be present in the skin when the hair growing on them is white, and when the hair is pigmented the underlying skin always appears to be also pigmented, though not necessarily the same colour. The colour of the pigment on the pads of the feet is usually similar to that of the nose. It has been suggested (Barnes and Barnes, 1946) that in parti-coloured Cocker Spaniels the pads are completely coloured by four weeks old in those which will become roaned, and spotted in those which will have clear white markings, but this is not always correct. The same authors point out that the skin of the eye-rims is always pigmented in dogs which are roaned.

The black pigmentation of the tongue of Chows is incompletely dominant in crosses with other breeds, the hybrids having coloured or partly-coloured tongues. The presence of pigmentation on the tongue is a fairly reliable indication of Chow ancestry in a mongrel.

There is no obvious mode of inheritance of toe-nail colour, which may vary from toe to toe in the same dog, but they are usually pigmented unless the toe is white, when the nail may be either white or coloured.

In the next chapter we will endeavour, in the light of the knowledge summarised here, to indicate the genetic formulae of various breeds and offer a little guidance on colour breeding. The interpretation used will be the classical one of the Extension, Agouti, and Albino series. Undoubtedly exceptions and anomalies will occur. A number of possible explanations of these can be offered: they may be due to mutations, abnormal conditions during development, reversal of dominance due to an unusual gene complex or to specific modifying genes, etc., etc. The situation would have become much clearer had more workers paid attention to the embryology, biochemistry, and microscopy of pigment in dogs, i.e. to the question of when and how it is formed, and to the size and arrangement of pigment granules in dogs of various colours and breeds. This, together with data on colour inheritance within breeds, would provide a firm foundation for carefully planned experiments involving breed crosses, with a view to revealing the different effects of the various 'colour genes' in different genetic backgrounds. Eventually a more coherent theory of the inheritance of pigmentation will have to be formulated, based on greater knowledge and a more physiological approach. However, the fact remains that the system here presented does give satisfactory guidance as to the outcome of the vast majority of colour crosses practised by breeders. A search of the genetic literature reveals only four instances which cannot be accounted for by the theory used here. They are:

1. Irish Red Setters throwing blacks as well as reds (Marchlewski, 1930);
2. Black-and-tan hound × agouti coyote throwing all black-and-tan pups (but there were only two, and the coyote could conceivably have been heterozygous $a^g a^t$) (Dice, 1942);
3. Two brindle Great Danes throwing blacks among their offspring (Marchlewski, 1947);
4. Two wolf-greys (agouti) throwing blacks (Marchlewski, 1930).

A few other examples, such as the Irish Setter × Lemon Belton English Setter litter which included black puppies (Whitney, 1948), could be accounted for by a rather improbable genic situation, but still within the system. As a last resort mutation can, of course, always be invoked.

Information from breeders clearly indicates that very occasionally Red Irish Setters do throw black-and-tan, plain black, and shaded sable pups, and Gordon Setters quite commonly throw 'Irish-coloured' reds. It would be very instructive if the coat colour of the Irish Setter could be analysed by crossing with breeds of known genotype in regard to colour, e.g. to sable Collies, yellow Labradors or Cockers, and liver Labradors or Cockers. It is possible that some breeds described as having Extension yellow ee may really be of the sable a^ya^y type; this may be suspected in any breed which commonly has black or chocolate hairs intermingled with the yellow along the spine and on the ears.

In closing this complicated and somewhat unsatisfactory section on the inheritance of pigmentation, it is appropriate to enlarge upon the suggestions of Winge (1950) that breeders could help scientists (and themselves!) by adopting a uniform terminology for describing coat colours. It would be most valuable if an atlas of coat colours could be published, using colour-photographs and naming the colours in a manner agreed on by interested bodies such as the Kennel Club and the National Coursing Club. Breeders could then refer to this atlas for the correct colour-description when registering their dogs.

VII

THE USE OF GENETIC FORMULAE IN COLOUR BREEDING

COLOUR breeding is a subject that has always attracted the more enterprising dog breeders. On the other hand, there are breeders who condemn colour-breeding as leading to the sacrifice of more important points to the quest for some rare colour. In working dogs, except in so far as colour directly affects their work, there is perhaps little to justify selection by coat colour, but in all breeds kept primarily as pets and for show, attractive coat colour is a great advantage. In some breeds, such as the Irish Red Setter or Manchester Terrier, only one coat colour is permitted and it is a sort of hall-mark of the breed. In others, such as Pekingese, Poodles and Cocker Spaniels, breeders have encouraged a wide variety of coat colour, which adds interest to breeding, and provides colours to suit all tastes.

The easiest coat colour to breed is one depending on a single recessive gene. Thus it is relatively easy to establish strains which will breed true for chocolate or for blue, but much more difficult to establish a true-breeding strain of wolf-grey, sable or black. Yet it is the latter colours that appear at first to be the easiest to breed, since being dominant they are not readily lost by mating to other colours. The phenomena of gene interaction and epistasis play such a big part in colour breeding that some understanding of them (Chapter I) is essential if complex coat colour situations are to be unravelled.

One simple example of the use of genetics in colour breeding must suffice to illustrate the possibilities. Suppose some enterprising breeder wished to introduce yet another new coat colour to English Cocker Spaniels. Blue-dilute Cockers could readily be produced by introducing the gene d through crossbreeding with any type that carries it. A breed whose coat-type was dominant to that of the Cocker would be preferable, as it could most easily be bred out. A blue Whippet might be a suitable choice. Mated to a black Cocker the progeny would probably be black, though reds or fawns might occur. They would all be heterozygous for the blue-dilution gene d. Mated again to black Cockers, half the progeny would carry d and half would not. The best thing would be to test-mate them by mating to a blue Whippet, discarding the resulting puppies and retaining for breeding those of the backcross parents which threw blues among their offspring by the Whippet. They could be mated to each other, and the blue puppies which resulted would be mated to pure Cockers and blues again extracted, by which time Cocker type would probably have been restored and all trace of Whippet, except the blue colour, lost. By this method, no further Whippet 'blood' would be introduced after the original crossbred generation (F_1).

This suggestion of crossing with another breed will of course rather shock the pedigree purists. It was, however, used during the formative period of almost every breed and it is entirely unscientific to refuse to utilise the simplest method of attaining what is required. The important thing is to know exactly what is wanted from the crossing and to make only such matings as are necessary to attain that end.

A similar method could be used to re-introduce black and other self-colours to Pointers in England. In this case a racy type of black Labrador would be a good source of the gene S. Since, however, self-coloured Pointers exist in other countries, the better method would be to import one. The colour is unimportant provided it is self-coloured, since, for example, a self-lemon mated to a black-and-white would throw self-black offspring (though they would probably have white on the feet, chest and face).

The remainder of this chapter will be devoted to examples of the genetic formulae of various coat-colours, with examples from several breeds, and notes on the results to be expected from matings between dogs of various coat colours. It is evident that the possible combinations of coat colour factors are almost infinite, but fortunately most breeds are homozygous for several factors, and contain only two or three alleles of others, and this considerably simplifies genetic prediction.

SOME EXAMPLES OF THE FORMULAE OF COMMON COAT COLOURS

(*a*) *Self-Coloured*

All self-coloured dogs must have at least one dominant allele of recessive white, be homozygous for the alleles of dominant white and of merle, and not be homozygous for complete albinism. In respect of these their genetic formula must therefore always include S–ww mmC–, or c^b or c^r.

The colour of self-coloured dogs may be:

Colour	*Formula*				
Black	E-	A-	B-	D-	
Chocolate (liver)	E-	A-	bb	D-	
Black-dilute ('blue')	E-	A-	B-	dd	
Chocolate-dilute ('apricot')	E-	A-	bb	dd	
Sable - black	E-	a^y-	B-	D-	
„ - chocolate	E-	a^y-	bb	D-	
„ - apricot	E-	a^y-	bb	dd	
Yellow with black or rusty nose ('Yellow' includes colours known as red, fawn, golden, etc.)	ee	**	B-	D-	
Yellow with liver nose	ee	**	bb	D-	
Wolf-grey (agouti)	E-	a^g-	B-	D-	
Bicolour *a.* black-and-tan	E-	a^ta^t	B-	D-	If a^s replaces one or both a^t the tan markings will be more extensive.
b. blue-and-tan	E-	a^ta^t	B-	dd	
c. liver-and-tan	E-	a^ta^t	bb	D-	

** any two alleles of the agouti series

(*b*) *Parti-Coloured*

The colour of the pigmented parts is determined in exactly the same way as in self-coloured dogs; any of the above formulae may therefore apply, but the animal must be homozygous for either white-spotting, ss, or the intermediate form s′– instead of S (but see discussion in previous chapter). It should be noted that when a bicoloured animal has also an extreme degree of white spotting the bicolour pattern may be obscured; thus in a parti-coloured saddle-marked dog which has coloured markings only on the ears these markings would be tan, and it could not be distinguished from a yellow-and-white or sable-and-white. The white areas will either be clear (rr), ticked (r^t–) or roaned (R–). Thus a black-nosed orange-roan Cocker has the formula (in full):

ss ww mm C– ee B– D– R– A– (or a^ta^t)

(*c*) *White*

White dogs must carry genes at every locus and may therefore have any of the above formulae, with any or all of the following factors which are responsible for the totally white coat:

W-	dominant white
MM	homozygous merle
c^dc^d	albino (with pigmented nose and eyes; there may also be an allele (cc) totally suppressing pigment, but its existence is uncertain).

None of these whites can be affected by roaning as they all entirely prevent pigment formation in the dog. Slightly coloured cream or greyish ('cornaz') animals have one of the higher alleles of c, *i.e.* c^r or c^b.

EXPECTED RESULTS OF SOME COLOUR CROSSES

Black to Black	will throw a majority of black puppies, but can throw any other colour except merle.
Liver to Liver	Majority liver. Can throw any other colour except black or blue or merle; in patterned coats such as bicolour, sable and agouti the black hairs will be replaced by chocolate ones throughout.
Blue to Blue	Majority blue; can throw any other colour except black or merle; but all pigment is affected giving blue-brindles, blue-fawns, dusky sables, etc.
Sable to Sable	Majority sable; usually a substantial minority of black-and-tans. *Note:* sables homozygous for chocolate may appear red with chocolate noses (chocolate-sables).
Yellow to Yellow ('extension yellow'ee)	Yellows only, or yellow and white; cream or recessive white without markings may be bred from yellows in certain breeds.
Sable to Yellow	All puppies black unless either parent carries both sable and yellow. Theoretically liver, blue and parti-coloured could occasionally occur.
Saddle (or bicolour) to Saddle (,, ,,)	All saddle or bicolour marked pups (but in any yellow pups the tan markings may be indistinguishable).
Merle to Merle	50% of the offspring merle; 25% black; 25% white, deaf, and blind. *Note:* Merle may affect animals basically blue, bicolour or liver, etc., in which case these will occur instead of black, and the shade of merle will be different.

Brindle to Brindle	**Mostly brindles, minority fawn. Can in certain crosses throw liver-brindle, black-and-brindle (bicolour), blue-brindle, etc.**
Agouti to Agouti	**Majority agouti, can also throw sable, saddle and bicolour, but not plain black.**
Agouti to Black	**Majority black; can throw any other colour of the agouti series.**
Liver to Blue	**Offspring black, unless either parent carries the other colour (blue or liver) recessively, in which case some 'apricot' (liver plus blue) may result.**
Liver to Yellow (black-nosed)	**Majority black; some may throw yellows with black or liver noses, or livers.**

Any of the above colours may throw recessive white or white marked (parti-coloured) pups. Inheritance of the colour of pigmented areas on parti-colours is the same as in the above examples. Two whites, if genetically different types, may throw coloured or partly coloured pups; this seldom occurs in matings within a breed but is common in crossbreds.

The following list indicates the coat colour formulae occurring in a few popular breeds:

Greyhounds, Boxers, Great Danes (probably also Whippets, Mastiffs, Irish Wolfhounds, Deerhounds, Bullmastiffs)	Homozygous AA BB ww CC rr, all of which can therefore be ignored. Boxers are also homozygous mm and DD, and Greyhounds mm. Colour in these breeds is therefore in effect determined by: Great Danes: E, e^b, e; M, m; D, d. Possibly S and s', affecting presence or absence of white on feet, etc., of non-harlequins. Greyhounds: E, e^b, e; D, d; S, s', s. Boxers: e^b, e (the dominant E has been almost bred out); S or $s's'$. Whippets: probably same as Greyhounds, but sometimes bicolour a^ta^t instead of AA.
Foxhounds, Bassets, Beagles, Bloodhounds, Harriers	Homozygous ww CC DD mm. Bloodhounds SS, others s'- or ss. Majority a^s- or a^ta^t (bicolour). Red, lemon or tan hounds may be of several formulae according to whether the yellow colour is due to extension yellow ee, sable a^y-, or extreme white-spotted bicolour. They may be B- or bb (liver) and the white markings may be clear (rr) or ticked. Ignoring those genes which are always homozygous, hound colours may have the following formulae:

Colour	*Formula*
Tricolour: black-clear white-tan)	E- a^s- B- rr or E- a^ta^t B- rr
black-ticked white-tan	E- a^s- B- r^t- or E- a^ta^t B- r^t-
liver-white-tan	E- a^s- bb rr etc.
Black-and-tan or liver-and-tan	E- a^s- B- r^t- (or rr)S-; E- a^s- bb r^t- (or rr)S- E- a^ta^t B- r^t- (or rr)S-; E- a^tt B- r^t- (or rr)S-
Tan(red)-and-white or lemon-and-white	ee a^s- B- r^t- ss ee a^ta^t bb rr ss E- a^y- B- r^t- ss etc. ee a^y- B- r^t- ss etc. E- a^s- B- r^t- ss with extreme white spotting obscuring the black saddle
Fox Terriers	Similar, but may be A instead of a^s or a^t, giving black-and-whites without tan.
Alsatians	Homozygous SS mm, usually EE. Colour normally depends on which alleles of the agouti series are present: A, a^g, a^y, a^s or a^t are all common in the breed. Occasional recessive white c^dc^d, cream (probably c^r), blue-dilute dd, and brindle and possibly rare 'extension' yellows ee. See also discussion of colour paling, p. 60.
Poodles	Probably the whites are c^dc^d but some may be extreme ss; solids are SS. The breed is homozygous ww mm rr and colour variations depend on the following: E or e (no brindles occur) D or d B or b C or c^d, possibly c^r. A or a^t (no other agouti alleles occur)
Cocker Spaniels	Normally homozygous mm ww DD (blue-dilutes dd occur in American Cocker, but not in the English breed). They are either A- or a^ta^t (a sable which, despite his statement to the contrary, bred as a^y- has been reported in American Cockers by Phillips (1938)). Formulae of common colours are as follows:

Solid	Black	E-	S-	A-	B-	R-	(or r^t or rr)	If a^ta^t replaces A- the dog is bicolour
	Liver	E-	S-	A-	bb	R-	,, ,,	
	Red or Gold	ee	S-	A-	B-	R-	,, ,,	
	Liver-nosed gold	ee	S-	A-	bb	R-	,, ,,	

Parti-colour	Same as solids but ss instead of S-. Roans are RR, 'clear whites' rr, and intermediate degrees of ticking r^tr^t or Rr^t.

Dachshunds	Normally homozygous SS ww CC EE DD, but rarely brindles e^be^b occur. Reds are a^y not ee. The gene A seldom or never occurs, all dachshunds being either a^ya^y, a^ya^t, or a^ta^t; and either BB, Bb or bb. Reds with bb are really chocolate-sables. Dapples have the gene M, as in merle collies.
Bull Terriers	Coat colour inheritance in this breed has never been satisfactorily elucidated, although there have been many attempts to do so. White is a simple recessive to coloured and is probably extreme white-spotting, ss. Whites seldom carry brindle, being genetically either red or black-and-tan as is sometimes revealed by small coloured markings. Brindle is reported to be dominant both to red and to black-and-tan. Darling (1932) suggests that the brindle is a heterozygous intermediate condition between red and black and cannot breed true, but data given by Grahame and Ritchie in the same book do not support him. Probably both types of red, a^y and ee, occur, as well as ee reds and brindle e^b.

The distribution of the two types of yellow-red colour between breeds is summarised below, as far as it is known.

Breeds having the Agouti Series Yellow, Sable, a^v *only*

Dachshund, Irish Terrier, Show Collie, Griffon Bruxellois, probably Border Terrier, Norwich Terrier, Finnish Spitz, Corgi, Dandie Dinmont, Cairn, and Basenji.

Breeds having the Extension Series Yellow e only

Greyhounds and probably all related types, such as Salukis, Afghans, Whippets, Deerhounds, Irish Wolfhounds, Borzois, etc.
Great Danes, Boxers, and probably Mastiffs, Bullmastiffs and Staffordshire Bull Terriers; possibly Bulldogs.
Labradors (yellow), Spaniels.
Probably Pomeranians and Pugs.

Breeds possibly containing both a^v *and e*

Bull Terriers
Hounds other than Greyhound group
Pekingese
Setters and Pointers
Golden Retrievers
Border Collies (may have only a^y)
Chows

VIII

SUCCESSFUL BREEDING SYSTEMS

ALMOST all the work reviewed in the preceding chapters may be described as observational rather than practical, in the sense that the object of the investigators concerned was to observe the manner of inheritance of various characteristics, improvement of their breeding stock being at most only a secondary consideration. Because of this, there has been a strong tendency to concentrate attention on those characters which appeared to follow the Mendelian manner of inheritance, other characters being dismissed as too complicated or too much influenced by environment to be 'suitable for study'. The practical dog breeder cannot run away from his problems in this manner, and if genetical science is really to be of more than occasional assistance to the breeder, these more difficult questions must be tackled.

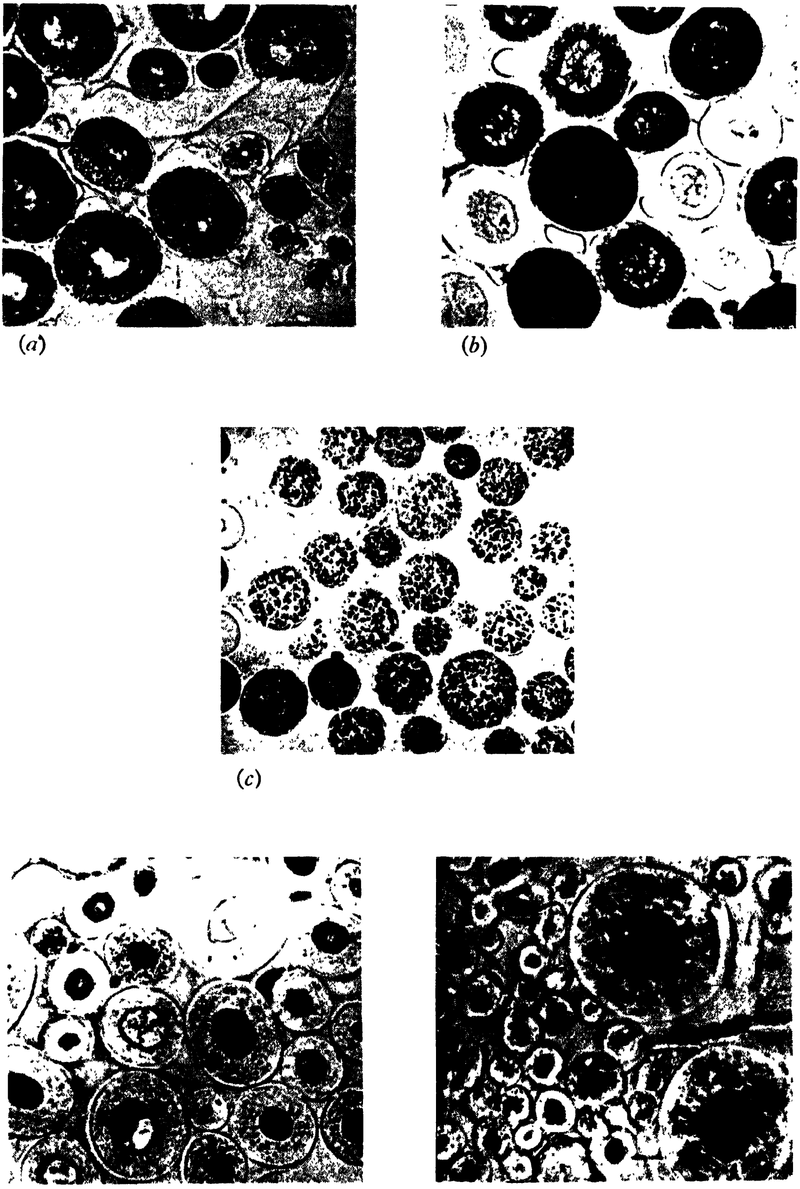
As stated on p. 9 only two reports are available of strains of dogs which have been deliberately established and developed under scientific guidance, but these two reports are of absorbing interest.

The two projects resembled each other in many ways. In both, the breed concerned was a working breed and success at work was the essential criterion by which the dogs were judged. In one case, that of Dr. Kelley's Boveagh strain of Border Collies (1947), one type of work only was required of the dogs, namely shepherding. In the other example, the Fortunate Fields German Shepherd Dogs (Alsatians) each dog was tested for suitability in a number of vocations and trained for that work for which it showed most aptitude. It is not possible to give a full account here of this work, but the remarkable book by Humphrey and Warner (1934) should be studied by every serious breeder, whether of Alsatians or other breeds. Unfortunately, this book is not at present readily available in Britain, and we are here confined to the discussion of its more strictly genetical aspects. The authors, although primarily concerned with the breeding of outstanding working dogs, gave considerable attention to the relationship between conformation, show points and working ability. Some of their conclusions, as to the inheritance of particular attributes, have been mentioned in previous chapters, and it is their breeding system with which we are now concerned.

It is a very significant fact that, neither in the case of the Boveagh dogs nor of the Fortunate Fields dogs, did the geneticists concerned start with any exact knowledge of the mode of inheritance of particular characteristics in their dogs. In both experiments, after many years' work, the authors express the opinion that certain characteristics are probably inherited in a simple Mendelian manner, either as dominant or recessive factors, but even in these few instances they do not claim that the mode of inheritance has been proved with certainty. In the meantime, before even that knowledge of Mendelian factors became available to them, the main object of their endeavours had been achieved as they had in fact succeeded in developing superior strains of dogs. They have done this far more rapidly and with a far greater degree of success than is usually attained even by the above-average dog breeder, indeed very few breeders have ever equalled their achievements. Clearly their success has not been based on simple Mendelism; on what, then, has it been based? The answer to this question is made very clear in both reports. Careful recording, objective analysis and assessment of the characteristics and performance of every dog, rigid selection of breeding animals on the basis of performance, pedigree, and where possible, some kind of progeny test; then linebreeding to animals proven as outstanding breeders; these have been the bases of success both in the Boveagh and the Fortunate Fields dogs. There is nothing in these methods which is obscure or beyond the reach of any intelligent dog breeder. Genetical theory influenced the investigators mainly in two matters, namely, the emphasis on progeny testing as a method of distinguishing 'genotype' from 'phenotype', and the calculations of 'inbreeding coefficients'.

Calculation of inbreeding coefficients is based on the assumption that each animal receives one-half of its inheritance (genotype) from each parent. Whilst, even on orthodox genetical theory this assumption is not strictly accurate, it is a much better approxi-

PLATE IX



(a) (b) (c) (d) (e)

Photographs by courtesy of the Wool Industries Research Association, Leeds mation than Galton's theory which asserted that each parent contributed one-quarter, each grandparent one-eighth and so on, or the Picardy system by which one-third of a dog's inheritance is assumed to be derived from its parents, grandparents and great-grandparents respectively. Obviously, everything a dog gets from its grandparents it must get *via* its parents, so that at the time of fertilisation the whole of its inheritance is derived from its parents, although not necessarily quite equally from each. The purpose of an inbreeding coefficient is to give a measure of the increase in homozygosity, i.e. the increased opportunity for the similar genes to reach the progeny through both parents.

Kelley found that certain matings, which would have resulted in pups with an inbreeding coefficient of more than 25%, were infertile or produced weakly puppies, although animals with a coefficient of about 20% were highly satisfactory. He therefore concluded that 20% of inbreeding was the maximum which could safely be used in this particular strain, and by calculating inbreeding coefficients in advance was able to avoid matings which would be dangerously close. No exact safety limit was established in the Fortunate Fields dogs, but inbreeding coefficients were used for guidance in a similar way. In both strains, a number of separate linebred families have been developed and then matings made between these inbred families. In the Boveagh dogs the families are all very closely related to each other and descended from the

PLATE IX. Pigment Granules in Dog Hairs. Cross-sections of dog-hairs, magnified × 400, showing size and arrangement of pigment granules. Photomicrographs by courtesy of the Wool Industries Research Association, Leeds. (*a*) Black hairs from a Border Collie. (*b*) Black and yellow hairs of various shades from an Alsatian. Note variation in size (diameter) of hairs. (*c*) Liver, and a few white hairs, from a liver-roan Cocker Spaniel. Note arrangement of the granules in clusters, and the even size of the granules, and of the hairs; also the absence of medulla (central hollow core) in the hairs. (*d*) Yellow hairs from a Golden Retriever, with pale yellow granules. The black centres shown in some of the hairs are due to air trapped in the medulla, not to pigment. (*e*) Blue hairs from a Greyhound. Note agglomerations of the black pigment in both cortex and medulla, in addition to the scattered granules.

same foundation animals, with a slight infusion of unrelated blood introduced from time to time to prevent too close inbreeding. This is the method of 'controlled heterosis'. The Fortunate Fields strain commenced with a larger number of foundation animals which were little related to each other, so that there is less danger of excessive inbreeding since the families within the strain are not so closely related as in the Boveagh dogs. In later generations, however, it will probably be necessary to introduce a trickle of outside blood.

Kelley, in describing the development of the Boveagh strain, does not give any analysis of the qualities required in his dogs, other than success at everyday shepherding work and at sheepdog trials; the only other quality mentioned is short coat, which is desired as it is considered more suitable than the rough coat for Australian conditions. The foundation animals were selected on account of their perfect temperaments and high working ability, and if any outcross is found to introduce any fault of temperament it and its descendants are immediately discarded from the strain. All pups are allowed to see and work sheep when about ten weeks old, before they know their names or have been controlled in any way, and pups for retention are selected at that time on the extent to which their instinctive behaviour is similar to that which is held as ideal for adults. This is probably the most important factor in selection. Attention is also paid to any special signs of intelligence, e.g. unusual ability to escape from the kennel enclosures.

Humphrey and Warner, on the other hand, give very great detail concerning the qualities required, and developed a scoring system by which the assessment of each dog was expressed. In all, 32 traits were listed for each dog, of which the great majority were concerned with structural characters or other physical attributes such as colour. However, the single trait, temperament, was given such a high proportion of the total score that no animal failing in this respect would be passed as a breeding animal. The only qualities penalised under temperament were shyness and distrust (see Chapter V). Other psychological traits such as intelligence, willingness, aggressiveness, etc., were not assessed in the total score, but were scored separately, since it was not considered desirable that dogs having a low score in these traits should be eliminated as breeding animals. Breeding animals were selected and paired with each other entirely on the basis of calculations (the formulae for which are given in their book) derived from the detailed records and scoring system. The main pressure of selection was against bad temperaments (shyness) and was successful in that there was a rapid drop in the proportion of shy dogs produced after the first generation in the strain. Traits which showed an upward trend (improvement) included intelligence, olfactory acuity, willingness, energy, and aggressiveness, whilst fighting instinct was reduced by selection against it. In the foundation animals there was a negative correlation between show points and good temperament, but this was reversed in the third generation when a positive correlation appeared. A higher proportion of successful workers was produced in later generations.

Several interesting correlations were revealed by analysis of the accumulated data. There was a definite positive correlation between light eyes and high intelligence, despite selection in favour of dark eyes. Chest development (depth and fore-chest) were correlated with good health, and so was flank development. Distrust was correlated with good tail-set.

There is much more of great interest in the book, but sufficient has been given to illustrate the results which were obtained.

Examination of the blood lines of any well-established breed reveals the fact that successful dogs have almost always been produced by linebreeding to famous animals. Thus every Border Collie that has ever been Champion at the International Sheep Dog Trials (Britain) has been descended, through Herdman's Tommy, from Old Hemp (McCulloch, 1946). Pardoe (1949) shows that 120 out of 148 champion Wire-Haired Fox Terriers are descended from Ch. Cackler of Notts, whilst in Smooth Fox Terriers 62 champions can be traced through Ch. Oxonian to Ch. Splinter.

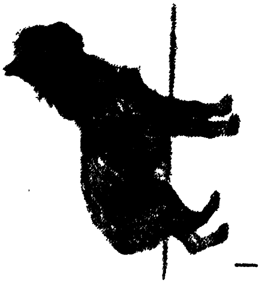
Haynes (1913) demonstrates the efficiency of linebreeding from the Airedale Terrier breed, and also shows how champions not belonging to the prepotent and successful line, failed to breed champions among their descendants. The 16 champions which between them sired 50 championship winners all trace back to the dog, Cholmondeley Briar.

It should be noted, however, that in some cases a famous stud dog was so widely used in his day that in subsequent generations it becomes difficult to find an individual which does not trace back to him, and this is true of the average and poor members of the breed as well as the winners. In such a case the dog can hardly be said to have founded the winning strains, though he may, like 'Obo' in Cocker Spaniels, have practically founded the breed.

Prepotency, although usually a characteristic of somewhat inbred strains, does not occur equally in all members of a strain or family. Some individuals have a strong tendency to reproduce their general type (Haynes, 1914) or to breed outstanding offspring to many different mates. Dogs and bitches with this tendency are extremely valuable to their breed. The prepotency of a breeding animal may be due to several causes: it may carry dominant genes for a few conspicuous characteristics, such as the black colour, short coat, and ear type which make the black Labrador predominant in most crosses involving this breed; there may be a general resemblance of the offspring to one parent in some physiological trait, such as the disease resistance of the Bull Terrier, and this is generally associated with a resemblance in type; prepotency in so-called quantitative characters is generally the result of inbreeding or, in breed crosses, of one breed having been established longer than the other. Environmental conditions during development may favour the exhibition in the offspring of qualities characteristic of one parent rather than of the other.

PLATE X. Type of Mating Called a 'Backcross'. Results of a cross between the achondroplasic Dachshund and the midget Brussels Griffon. 1. Brussels Griffon ♀. 2. Dachshund ♀. 3. F_1 ♂. 4-7. Backcross of F_1 with Dachshund. 4. ♀. 5. ♂ (L.) and ♀ (R.). 6. ♂ (L.) and ♀ (R.). 7. ♀.

♀ = female; ♂ = male



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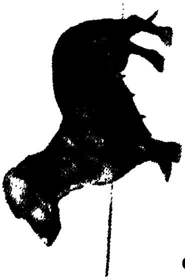

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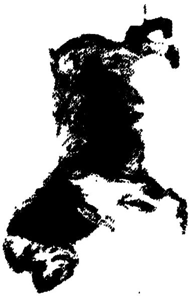

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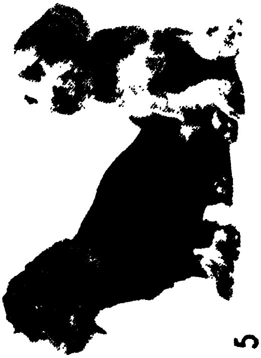

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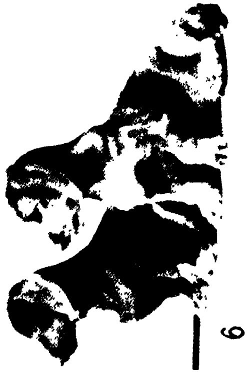

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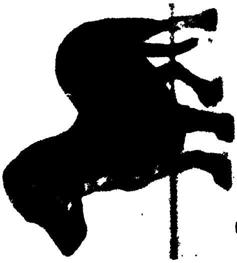

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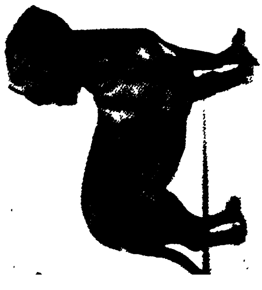


PLATE XI (*a*)

(*b*)

IX

THEORY AND PRACTICE

We have now completed our review of scientific studies of inheritance in the dog. Despite the large number of investigations reported, and the very many interesting and useful facts which they reveal, they cannot be considered to comprise a really satisfactory picture of the genetics of the dog; it includes too many conflicting statements, too much laborious presentation of trivial facts, and too little serious endeavour to improve the dogs. This arises very largely from the fact that, with few exceptions, the scientists have not been practical dog breeders and the practical dog breeders have not been scientific. When science and practice are properly combined the results are very gratifying, as instanced in the work of Kelley, of Humphrey and Warner, and, to a less extent, of Whitney. A contributory cause of this divorce between theory and practice has undoubtedly been the narrow and restricted approach of the majority of scientists who concerned themselves with dogs. Perusal of the literature of canine genetics clearly indicates that the search for Mendelian factors, whilst yielding useful information, is not an adequate form of investigation. Such knowledge has in practice been but little used, even by those scientists who founded successful strains of dogs.

The majority of characters of major importance in dog breeding are included in the category usually called 'quantitative characters'. By this term is meant a characteristic or trait such as size, speed,

Plate XI. Effects of Selective Breeding. The best of the modern Cocker Spaniels are nearer to the ideal described in the 'Standard of Points' than were any Cockers existing at the time the Standard was originally drawn up; selective breeding has successfully changed the type in the direction desired by breeders, giving shorter backs, deeper muzzles, etc. (*a*) dog born in 1879 and his daughter; (*b*) bitch born in 1945.

or intelligence, which every dog has in some degree, but which varies in amount or quality. Such characters are assumed to depend on the interaction of a very large number of genes scattered through all the chromosomes, and each able to supplement or reduce the effects of the others. Within a breed the range of variation is limited because by selection a certain collection of such genes has been gathered together, and although they are still so numerous and varied that no two animals are likely to get exactly the same set, yet the variation is much less than that which occurs in a litter of crossbreds. No simple dominant/recessive relationship occurs, and breeding methods must aim at restricting the range of variation or 'shifting the average'. This will be discussed more fully below.

The inheritance of 'quantitative characters' has not received detailed attention in dogs, but much work on problems of this kind has been carried out on farm livestock, such as poultry and dairy cows. The improvement of a breed in respect of such characters depends, not on the attempted identification of individual genes, but on the measurement of the 'quantity', e.g. milk yield, in various individuals and groups, and the study of how it varies in the population as a whole, and in that part of a population which has been bred in a particular way, such as in a certain strain, or among the offspring of a certain sire. The identification of a particular 'Mendelising' gene in one kind of animal, or even in one breed, does not imply that this gene necessarily exists in other types of animal, and each must be investigated separately (although genes acting in a similar manner have been found in many animals, e.g. the coat colour 'blue dilution' is known in mice, rabbits and cats, as well as in dogs). When quantitative characters are being considered, however, the results found in one type of animal can be applied rather more directly to another type, since no identification of genes is involved. Research in quantitative characters has led to the formulation of practical breeding methods based on accurate assessment of performance, whereas research on 'Mendelism' has tended to lead to observational studies rather than to active breed improvement. Rather complicated statistical methods are used in the former kind of work, and only exceptionally mathematical dog breeders would attempt to use them fully. Interested readers may pursue the matter further by studying textbooks on livestock breeding such as that of Lush (1945), but, for the majority, the information included in the latter part of the present chapter will provide a working knowledge of the guiding principles.

One of the difficulties in dealing with 'quantitative' characters is that their expression is always much influenced by the environment.

The prime concern of dog breeders is with the adult dog or bitch; the appearance or performance of the puppy is of little significance except as an indication of what it will be like at maturity. The appearance and performance of the adult animal are, however, the outcome of many influences. Every breeder of show dogs knows how easily a promising puppy can be ruined by bad rearing, and even more evident is the influence of training on the performance of working dogs.

The most controversial question in all the history of the science of genetics has been whether or not peculiarities acquired by an individual during its lifetime can be passed on to its descendants. According to the Mendelian theory of genetics, this is impossible because the sex cells are held to be direct descendants of the sex cells of previous generations, and to be insulated from any adaptive changes which may take place in the rest of the body, the 'soma'. This view, based on the results of experimental crossbreeding and observation of the behaviour of chromosomes during cell division, received support from many experiments in which controlled attempts to achieve the inheritance of acquired characters failed. However, practical livestock breeders were never entirely convinced that acquired characters could not be inherited, and the belief that they can be is particularly widespread amongst those interested in training working dogs. It is therefore of special interest to dog breeders that the whole question has been reopened in scientific circles in recent years by what has become known as the 'Lysenko controversy'. Lysenko, a Russian scientist, basing his work on that of a famous horticulturist called Michurin, has formulated a new genetical theory opposed to Mendelism. He maintains that since the germ cells take part in the processes of chemical exchange (metabolism) which are characteristic of all living things, it cannot be true that they are never affected by changes in the rest of the body and in the environment; that the germ cells are part of the body and not merely mechanically enclosed and protected by the 'soma'; and that heredity is not a special function carried out by special organs (the chromosomes) but is 'the property of an organism to require certain conditions for its development' which is determined for it by its parentage, but may be modified by unusual conditions of development, in which case it may give to its offspring qualities not derived from its own ancestors. Although Lysenko's ideas have been condemned, on the evidence of their experiments, by the great majority of geneticists outside the Soviet Union and the 'New Democracies', they have not been without support from a few geneticists and other biologists in the 'West', and have aroused much interest among farmers. It is claimed that Lysenko's breeding methods have greatly assisted farming in those countries in which they have been applied.

Lysenko particularly emphasises that there is no question of any and every acquired modification being inherited, but believes that certain physiological adaptations can sometimes be passed on, if the causal influences affected the animal during a critical phase of its development. He considers that scientists should endeavour to discover these critical 'plastic phases' in order to influence them, and that the result of such influence is not likely to develop gradually, but to take the form of a sudden conspicuous change after several generations have been treated without apparent effect. It is to be expected, however, that heredity will tend to be strongly conservative and not respond easily to improved conditions, especially in old-established breeds or strains, or those in which heredity has been stabilised by inbreeding. This is especially true of mammals, in which the foetus is so well protected from the outside environment during the early and probably crucial stages of development.

The orthodox theory that inheritance is entirely determined, at the time of fertilisation, by the genes which the fertilised egg happens to contain, is in some respects illogical. The genes are believed to act somewhat like chemical catalysts, or enzymes, or to produce enzymes, substances which assist chemical changes to take place without being used up themselves. Thus a substance A may combine with another substance B only at very high temperatures or very slowly; but if a catalyst or enzyme C is added it makes the reaction possible at low temperatures, or speeds it up, by combining temporarily with A in a manner which enables B then to attach itself to A, whereupon C is released and can assist the process again. If genes do act in this kind of way, then obviously the rest of the living substance or its food must play the part of A and B in the above example (technically the substances with which the enzyme reacts are called the 'substrate'). The enzyme may be said to control the reaction in the sense that it would not take place under those circumstances without the enzyme: but it would not take place under any circumstances without the substrate. Therefore, even if there are genes which produce enzymes to guide the course of embryonic development, the rest of the fertilised egg and its ability to make use of the available nutrients must also be regarded as a part of heredity, and perhaps provide the means by which physiological changes could become fixed in a strain. Mendelians regard only those inherited features which are already determined in the newly fertilised egg as being 'true heredity', any characteristics due, for example, to the effects of the mother during pregnancy and lactation being regarded as inborn but not inherited.

The methods of practical livestock breeders never give a really decisive answer to the question of whether or not acquired characters are inherited, because the practical method is always to select for further breeding those animals which respond best to the conditions under which they are required to live and work, and progress so made can be explained on either theory. This applies also to the work of Kelley and of Humphrey and Warner. Perhaps the wisest conclusion is that of Horlacher (1927) who, speaking of sheep, said: 'Breeders should of course continue to develop them to the highest degree of perfection, for if this development is cumulative from generation to generation, then the profit certainly will be great; but even if the development is not cumulative, the breeder can determine which animals are inherently capable of responding to favourable environmental conditions.' Thus breeders may bear in mind that there is at least a possibility that environmental influences, especially those affecting the young puppy, may become heritable if repeated for many generations; according to Lysenko this is more likely to happen if the parents were unrelated or were reared in widely different environments, e.g. different countries. In this connexion it is interesting that dogs of British breeds (especially Terriers) exported to the American continent are said to 'degenerate in type' after a few generations (this is mentioned by Stockard, 1941), unless frequently replenished by matings with stock imported from Britain. The Mendelian explanation would be that climatic or other environmental influences had eliminated (by death of puppies, etc.) those genotypes which gave the desired 'type', as such genotypes happened to be unsuited to American conditions. However, as the breeders' selection of show stock is always in favour of the desired type, and the immediate offspring of imported stock are satisfactory in type, Lysenko's explanation of a direct effect of the environment on the heredity (after a few generations) is attractive.

There is also the possibility that conditions at first only tolerated may eventually become a necessity; there is some evidence that breeds subjected for generations to high feeding and generous vitamin supplements may be more subject to deficiency diseases than are the less cared-for breeds like Border Collies and Foxhounds. The possibility that the stultifying effect of kennel life may adversely affect inherited intelligence and working ability cannot be entirely excluded.

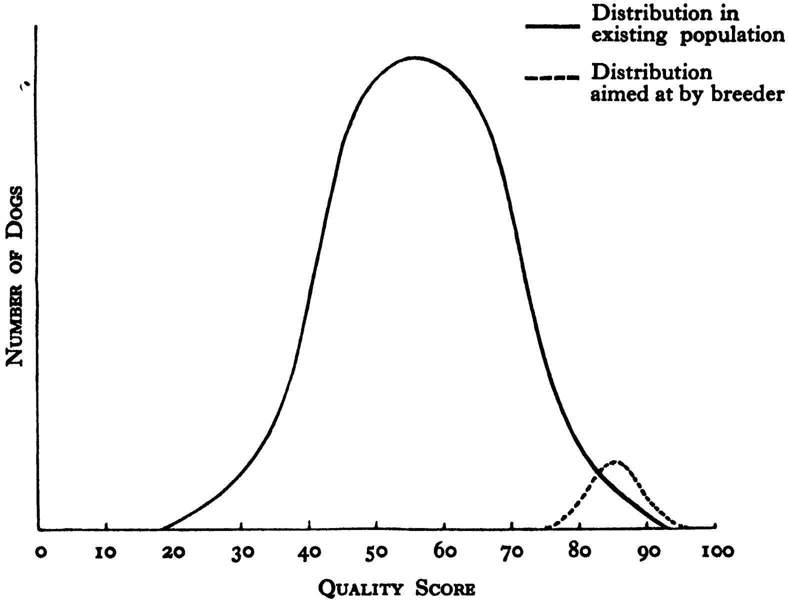
A rather different matter discussed by Lysenko is selective fertilisation. In common with several workers, of whom at least two (Pearson and Usher, 1929; Whitney, 1948) have been concerned with dogs, he considers that the union of a particular sperm (out of the millions available at a normal mating) with a particular ovum (egg-cell) is not a matter of chance but of selection. This could account, among other things, for the consistently higher percentage of male than female puppies born, and for certain failures to obtain Mendelian ratios. Selective death of embryos is an alternative explanation in most of the latter cases, but it cannot explain the inequality of the sex-ratio, and the possibility that fertilisation is not entirely random cannot be ruled out. Quite probably sperm of different heredities differ in activity, length of life in the female tract, etc., and statistical averages may at times conceal important individual differences, such as certain dogs or bitches regularly throwing more of one type than simple Mendelism would lead one to expect. Such individuals, if they exist, may be very important, especially if the type they throw is valuable and rare, but although such animals are often referred to by breeders, their breeding records have never been subjected to scientific analysis.

Reference has been made to pedigree as one of the bases of the work of Kelley and of Humphrey and Warner. However, the value of pedigrees in breeding is very widely misunderstood. Pedigrees issued by breeders or by the Kennel Club, etc., consist normally of a mere list of names, with nothing to indicate the quality or successes of the animals, except if they have won the title 'champion' or a working qualification. A pedigree, to be of real use for guidance in breeding, must clearly indicate the faults and virtues of every dog and bitch, together with any available information as to the quality of its progeny. Such a pedigree record requires a card index system rather than the usual brief sheet of paper (*see* Appendix I). Most breeders content themselves with carrying in their heads information about the dogs they have bred or which they have personally known and assessed. The majority of newcomers to a breed have little knowledge of the dogs in the pedigree of their foundation animals, and have great difficulty in extracting information even about living stud dogs to which they might send their bitches, unless the breeder is lucky enough to be able to found his kennel on a recorded strain.

Pedigree recording, correctly developed, represents a progeny test of the ancestors. In general, long pedigrees are of more interest to the historian than to the active breeder. An animal in the fifth ancestral generation (great-great-great-grandparent) contributes only about 3·125 per cent of the inheritance of a pup; if this includes some particular and important trait, such as a desired coat colour which is rare in the breed, such a distant ancestor may become important. However, for most purposes, a thorough knowledge of three or four generations of the pedigree is all that the breeder really requires. It is more important to extend this knowledge than to extend the pedigree. The breeder, having decided which traits are to receive most attention in selection or elimination, should endeavour to discover not only how each near ancestor was endowed in regard to these traits, but also how its litter brothers and sisters and its offspring were endowed. This information should be recorded in the pedigree index.

If at any time it was possible to assess the merits of every living individual in a breed, giving a score number to each dog or bitch according to its quality, it would be found that there was a range from bad to excellent, and if the scores were plotted as a graph, the result would probably approximate to a 'normal curve' (Fig. 3). The merit of animals produced by any dog breeder could be plotted similarly, and the aim should be to produce a strain whose merit-curve lies above (further to the right in Fig. 3) the curve for the breed as a whole. However, if success in competition is the aim, the curve to be considered for comparison is not that of the breed as a whole, but that of other competitors, and it is this level of merit which the successful strain must exceed. If the merits of two breeding animals place them both near one extreme of the curve—whether both are exceptionally good or exceptionally bad—their progeny will tend to be nearer to the average of the breed than the parents are, although the range of the litter will be towards the parents' end of the curve. If one parent is exceptionally good and the other bad, the tendency is for the range of

FIGURE III: Example of a normal Curve



merit of the litter to be similar to that of the breed or strain as a whole. A very mediocre bitch thus tends to produce some puppies better than herself even if her mate is equally mediocre, and if she is mated to a champion stud dog, not all the credit for the improved merit of the litter as compared to their dam is really due to the dog. Similarly, when two champions are mated together, they cannot be expected to produce a litter of puppies all as good as themselves, although the range of the litter should be at the top end of the merit curve, and the best pup as good as, or better than, the parents.

There is always a danger in talking in terms of tendencies and averages, that these will be taken as rigid and absolute. Part of the art of breeding lies in the selection of mates which will 'nick' with each other and produce offspring which revert little or not at all towards the average of the breed. Once the tendency or 'drag of the race' is recognised, the breeder judges his success by the range of merit in his puppies rather than by the production of a few brilliant individuals amongst a lot of duds. The importance of the *worst* puppy of each generation then becomes apparent: if the worst pups produced in the fourth generation of your strain are just as bad and just as numerous as the worst in your first generation you have made no real progress, even though you may have bred a champion. It is a good plan for the breeder of show dogs to give the worst pup in each litter to some neighbour as a pet, and to ask him to bring it to visit the kennels occasionally. Thus the breeder will not be allowed to forget its faults, and will have plenty of opportunity to compare it with the worst of later generations.

A scoring system, such as that used by Humphrey and Warner, to express the merits of each dog, is often very useful, especially where several people are concerned in developing the strain, or where so many animals are involved that personal knowledge of each is not possible. It is also a very useful addition to pedigree information, the recorded score of an ancestor being more durable than the breeder's memory. The only difficulty is to develop a scoring system suitable to the particular project, which involves detailed definition of the aims and the relative importance of each trait considered, and consistent methods of evaluation of animals scored. Another difficulty in ordinary breeding operations is to establish a criterion of success. The Fortunate Fields dogs were all given, as nearly as possible, equal opportunities in the hands of experienced trainers to develop and reveal their qualities. The ordinary breeder cannot give equal opportunities to his dogs, and this is particularly true when the objective of the breeder is to produce dogs which will win prizes in the show ring. Wins depend so much on such things as the quality of the other competitors, the whim of the judges, the skill in preparing and handling the exhibit; every breed has had inferior specimens which have become champions through luck or influence, and brilliant specimens which never attained great fame. These elements, which we may call 'luck' enter into every success which depends on competition, but competitions for working ability have one great advantage over 'beauty' competitions: a really bad worker will never win consistently in working tests, even if his owner can 'pull strings'. The breeder of show specimens must therefore rely primarily on his own judgment of each animal, whilst the breeder of working dogs can safely rely on competitive success as a criterion of the quality of a dog, even if he has never himself seen the animal at work.

Although the establishment of an above-average strain should be the objective of each breeder, commercial aspects of breeding exhibition dogs do not entirely encourage such a serious attitude. It is much more profitable to produce and sell one champion than half-a-dozen excellent dogs which just miss championship class. A good dog spoilt for show by one minor fault will sell for little more on the pet market than will a complete dud. Therefore, the common practice of purchasing a third-rate foundation bitch and sending her to the most popular champion stud dog of the moment is often justified commercially though not genetically: if one brilliant puppy results, the reputation of the breeder and even of his brood bitch will be established, even though all the other puppies

PLATE XII. Effects of Selective Breeding in contrast with Pl. XI. The Deerhound has remained essentially unchanged in type for nearly fifty years, selective breeding having in this breed been used, very successfully, to keep the original type constant. (*a*) dog born in the early years of the century; (*b*) bitch born 1945.

PLATE XII (a)

(b)

were hopeless specimens. This is the fault of commercialism in dog breeding and will continue as long as dogs are bought and sold for shows. The serious breeder will only benefit financially if he can afford a fairly heavy capital investment and wait some years until he begins to reap the benefit of the high average quality of his stock. It is also difficult or impossible to establish and maintain a strain if the breeder is unable to keep a large number of dogs. Probably six adults and a varying number of young stock is about the minimum, and even then it would be very desirable to put a few additional bitches out on breeding terms as a reserve in case of heavy losses at home. The great majority of those who make a hobby of dog showing cannot keep sufficient numbers to establish a strain of their own. There is great opportunity for co-operation among small breeders, or between them and a larger kennel with an established strain. An agreed breeding plan and recording system can be adopted, experiences shared and mutual assistance given, each owner taking pride in assisting to develop a high average merit in the strain as a whole. Through such co-operation the smaller breeders could gain the greater numbers of dogs and increased capital which give the big kennel so many advantages; and at the same time their dogs, being distributed in twos and threes among co-operators, would get the personal attention and companionship which is inevitably to some extent denied to the kennel dog. The British Kennel Club has recently ruled that dogs collectively owned, e.g. by breed clubs, may not compete for prizes at shows. The purpose of this ruling is not stated, but it certainly appears likely to discourage one type of collective effort at breed improvement.

The foregoing recommendation on breeding methods, based mainly on the work of Kelley and of Humphrey and Warner, can now be briefly summarised in the form of a breeding plan for the foundation of a strain:

(1) Decide on a few traits which are regarded as essential and on any faults considered intolerable. Whatever the breed, disease resistance, fertility, and absence of deformities, must be included as essentials, and certain character failings, such as viciousness, extreme nervousness, and hysterical or epileptic tendencies, must be condemned.

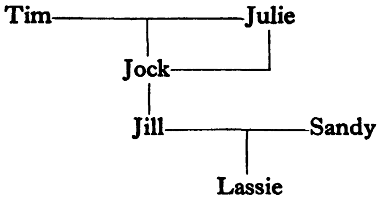
(2) Develop a scoring system in which the selected virtues and

faults receive marks in accordance with (*a*) their importance to your purpose or breeding aim, (*b*) their rarity or otherwise in the breed (or show population of a breed) as a whole. Virtues which are well-established in the breed and present in every individual used or bred need not be scored. As certain traits, rare at first, become established in the strain, their scoring may be reduced in order that greater weight and attention may be given to some other trait. Or the scoring system may remain the same, attention in matings being concentrated on scores for individual traits which require improvement.

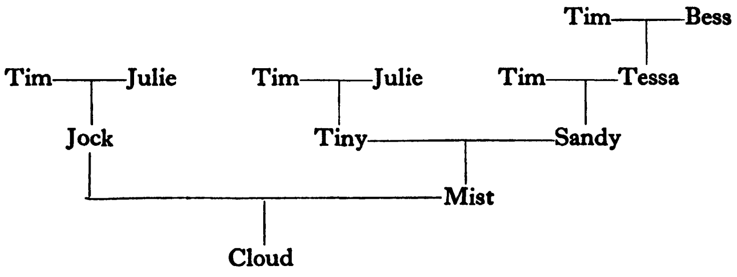
(3) Linebreed consistently to the best individual produced until a better one occurs, then linebreed to that. The blood of an outstanding dog or bitch can only be conserved by inbreeding to it whilst the animal is living, but close inbreeding should only be resorted to when an animal of very exceptional qualities and with no outstanding faults is available. If inbreeding results in unsatisfactory litters this does not condemn the favoured animal, but merely indicates that a less close mating should be made. Wide outcrosses should not be resorted to after the establishment of a strain, but some outside blood should be introduced, e.g. by the use of a dog sired by one of the strain from an unrelated bitch. In the foundation animals relationship need not be close, in fact wide outcrosses will give more variation and therefore greater possibility for selection of desirable combination of traits. Every animal to be used in the breeding programme must pass rigid assessments for individual excellence as well as average excellence of its relatives including its progeny when known.

In following such a linebreeding programme it becomes desirable to find a way of expressing the closeness of inbreeding and the amount of influence which any animal in the pedigree may be expected to have on the offspring of a particular mating. The 'Picardy system', well known to Alsatian breeders, is an attempt to express such relationships, but it tends to exaggerate the influence of the 'chosen' animal by allowing too little for the influence of its mates. The method commonly used by geneticists is Wright's Coefficient of Inbreeding (1923), but this involves quite a complicated calculation, and it does not give any absolute measure but only the percentage by which the homozygosity of the offspring is likely to exceed that of the breed or strain as a whole. For practical purposes a useful method is to calculate the so-called 'percentage of blood' on the assumption that each parent contributes one-half (50%) of the inheritance of each pup, each grandparent 25%, each great-grandparent 12·5%, each great-great-grandparent 6·25%, and so on. Thus, if the same dog sires both the sire and dam of a litter it contributes as much (50%) as if it had itself sired the pups. The pedigrees in Diagram 4 further illustrate this type of calculation.

Diagram 4



Pedigree 1 showing 37.5% 'blood relationship' of Lassie to Julie, but no inbreeding to her; the inbreeding which occurred in the son × dam mating is nullified by the complete outcross to Sandy, which is not descended from Julie.



Pedigree 2 showing 37.5% 'blood relationship' of Cloud to Julie, with inbreeding to Julie, and also linebreeding to Tim.

Two pedigrees, illustrating linebreeding, inbreeding, and 'percentage blood relationship'.

In addition to the percentage of blood, however, it is important to know how many times the same individual appears in a pedigree, and especially whether heredity derived from him (or her) can reach the offspring through both parents. If any individual occurs in the pedigree of both the sire and dam of a litter, inherited factors derived from that individual may reach some of the offspring through both parents (this is, indeed, the essence of inbreeding), but this is the less likely to happen, the further back the common ancestor is in the pedigree. If a certain individual occurs many times in the pedigree of one parent but not at all in the pedigree of the other, then the litter is not inbred, even though the 'percentage of blood' of that individual may be high, and his influence therefore greater than that of any other individual in the pedigree. The 'percentage of blood' cannot, however, exceed 50 without inbreeding.

The term 'linebreeding' is usually understood to mean building a pedigree in which one individual occurs repeatedly, but it is possible to linebreed to several individuals at once. It is not possible to linebreed without inbreeding to some extent, but it is possible to inbreed without linebreeding in this sense of the term, e.g. by mating brother to sister in every generation; some breeders, however, would call the latter linebreeding, on the grounds that the animals mated belong to the same 'bloodlines'.

In addition to the general method of linebreeding and recording outlined above, knowledge of those factors which do follow the simple Mendelian mode of inheritance can of course be utilised without departing from the general system. Thus, for example, a particular factor can be introduced from another strain if necessary, by the method outlined in the chapter on colour breeding. Such factors will be inherited in the manner described as 'simple Mendelian inheritance' (Chapter 1) whether there is inbreeding or not. The calculation of 'percentage of blood' or of 'inbreeding coefficients' is useful only where more complex modes of inheritance are involved, and simple Mendelian factors are not affected.

The better use of the accumulated knowledge of inheritance and of breeding methods will assist the serious breeder more rapidly and more certainly to approach his objective. The choice of objective is the responsibility of the breeder; if it includes deformities, weakness, and ill-temper they will as surely be obtained as were the splendid qualities of the dogs of Boveagh and of Fortunate Fields.

APPENDICES

APPENDIX I

NOTES ON CARD INDEX PEDIGREES

THOSE not familiar with the use of a card index may find the following examples of value; it should be remembered, however, that these are only examples, and each breeder will have to work out the details to suit the particular breeding stock and objective. Records of Show or Working Trial wins are probably more conveniently kept in a notebook owing to their possible bulk if kept on cards, but there should be a reference number on the individual's card to the book in which its wins are recorded.

PEDIGREE CARD

FRONT

Number and sex: 10b.

Dam: 1b Sire: 2d
Date of Birth: 15/7/43 in litter No. 1b/3
K.C. Registration Number:
Colour:
Quality Score:
Temperament Score:
Health Record:
Died (date): Cause of Death:
Health Score: Age at Death:

BACK OF CARD (BITCH)

Breeding Record—Bitch No. 10

Dates of Heats	*Dates Mated*	*Dog*	*Date Whelped*	*Litter No.*
9/3/44 to 28/3/44	—	—	—	—
30/9/44 to 20/10/44	11/10, 13/10	No. 4	—	—
2/4/45 to 23/4/45	12/4, 14/4	No. 4	15/6/45	10/1

BACK OF CARD (DOG)

Breeding Record—Dog No. 2

Dates Mated	*Bitch*	*Litter No.*	*Number of Pups*
12/5/43, 14/5	No. 1	1b/3	7

Litter Card

Date of Birth: 15/7/43 *Litter No.* 1/3
Type of Birth: (normal, prolonged, Caesarian, etc.)
Sire: No. 2 Dam: No. 1
Number of puppies: 7 Alive: 6 Stillborn: 1

Pedigree No.	*Sex*	*Colour*	*Weaned or Died*	*Notes*
5	d	black	stillborn	
6	d	black	weaned 10/9/43	
7	d	blue	weaned 10/9	
8	d	fawn	died 20/7	crushed by bitch
9	b	black	weaned 10/9	
10	b	fawn	weaned 10/9	
11	b	fawn	weaned 10/9	overshot; given away, no pedigree card kept.

Notes. To be effective, remarks on cards should be brief so that they can be seen at a glance. Explanations of scoring systems and any lengthy notes can be kept in reference books. It is a good idea to have a photograph of each dog, with date when taken, on the pedigree card or on a supplementary card; alternatively a photograph album may be kept with references to it on the cards. A card should be kept up to date, as far as possible, even for animals sold, unless they are definitely culled from the strain and disposed of on the understanding that they will not be used for breeding. Dogs and bitches not owned by the breeder, e.g. parents of the foundation animals and stud dogs to which bitches are sent, should have pedigree cards made out and filled up as far as the information available permits.

Appendix II

GLOSSARY

The following brief definitions of expressions occurring in the book are designed to assist the non-scientific reader; the genetical terms are further defined and discussed in the text, especially in Chapters I and IX.

Achondroplasia. A condition in which the limbs are abnormally short due to a disturbance in ossification of the cartilage of the long bones.

Alleles or Allelomorphs. Alternative forms of the same gene influencing the same developmental process or processes, but in different ways.

Chondrodystrophy. See Achondroplasia.

Chromosomes. Dark-staining bodies visible at cell division, which carry the factors for heritable characters, the genes. They occur in pairs, one derived from the father, the other from the mother. Members of a pair carry the same or allelomorphic genes in identical arrangement.

Cortex. The solid part of a hair.

Cryptorchidism or Cryptorchism. A developmental defect in which the testes (testicles) remain in the abdominal cavity instead of descending into the scrotum.

Dominant. Said of one of a pair of alleles whose effects are expressed to the exclusion of the effects of the other allele. Animals heterozygous (*q.v.*) for a dominant gene cannot be distinguished from animals homozygous for it except by test matings. In genetical notation a recessive gene is usually symbolised by a small letter and its dominant allele by the corresponding capital (*e.g.* D=gene for black coat colour and d=gene for blue).

Epistatic. Said of a gene or character which is expressed despite the presence of other, non-allelic, genes which tend to produce a different or contrary effect. Epistasis (n.) is similar to Dominance except that the relationship is between non-allelic genes.

F_1. The first generation offspring of a given mating. The F_2 (2nd generation) is produced by intercrossing the F_1.

Factor. The term originally used for what is now called gene.

Gene. The unit of heredity, believed to be the physical basis of inherited characteristics. A gene occupies a definite position on a definite chromosome.

Genetics. The science of heredity, variation, sex determination and related phenomena.

Genotype. The hereditary make-up of an individual.

Heterosis. The superiority over either or both parents of the progeny resulting from the crossing of strains, varieties, breeds or species. Known also as Hybrid Vigour.

Heterozygous. Not pure or true breeding for a given factor. Containing two different alleles of the same gene. A heterozygote produces two kinds of germ cells with respect to the gene in question.

Homozygous. Pure or true breeding for a given character. Having the gene for the character in duplicate, a homozygote produces only one kind of germ cell with respect to that gene.

Hybrid. (1) The offspring of two parents of unlike genetic make-up. (2) A heterozygote for one or more genes.

Hydrocephalus. Condition in which there is an abnormal increase of cerebral fluid; marked by enlargement of the head.

Hypostatic. Said of a gene or character which cannot be expressed in the presence of other, non-allelic, genes which tend to produce a different or contrary effect. The converse of Epistatic (*q.v.*)

Lethal. The name given to a gene which, when present in the homozygous condition, causes the death of the embryo. When the inherited condition is one which leads to premature death after birth, it is more correctly called sub- or semi-lethal.

Linkage. The tendency for two or more characters to be transmitted together, because the genes are located in the same chromosome.

Medulla. The central hollow core sometimes present within the cortex of a hair.

Modifiers. Genes which by themselves have no noticeable effects, but influence the effects of other genes present in the same organism.

Monorchidism or Monorchism. Condition in which one of the testes has failed to descend into the scrotum.

Monovular. Developed from one fertilised egg, as identical twins.

Nicking. When individuals produce better progeny when mated with some strains than with others, those that give rise to the superior progeny are said to have nicked. The phenomenon probably depends on the maximal complementary action of genes from one strain with those of the other.

Oestrual prolapse. Prolapse of the uterus tending to recur during each oestrus.

Oestrual weakness. Weak or incomplete manifestation of oestrus.

Oestrus. Heat, breeding 'season'.

P_1. The parent generation; P_2. grandparent generation, etc.

Phenotype. The appearance and/or performance of an individual, *i.e.* the outcome of the interaction between its genotype and its environment.

Prepotent. Said of an animal with an unusually strong tendency to pass its characteristics on to its offspring. Probably due to the presence of many dominant alleles in the prepotent parent.

Recessive (gene or character). A gene or character which is not expressed in the presence of its dominant allele.

Segregation. Manifested in the F_2 (and later hybrid generations) as a separation and distribution to different individuals of the Mendelian characters in which the parents of the F_1 hybrid differed.

Sex Linkage. Association of a hereditary character with sex due to the fact that its gene is on a sex chromosome.

Soma. The body, excluding the reproductive cells.

REFERENCES TO THE LITERATURE

[*A.B.A.*, . . .] indicates that the paper has been abstracted in *Animal Breeding Abstracts*.

AHMED, I. A. 1941. Cytological analysis of chromosome behaviour in three breeds of dogs. *Proc. roy. Soc. Edinb.*, *B*, *61*: 107-118. [*A.B.A.*, *10*: 51.]

ALLEN, G. M. 1914. Pattern development in mammals and birds. *Amer. Nat.*, *48*: 385-412; 467-484; 550-566.

ANKER, J. 1925. Die Vererbung der Haarfarbe beim Dachshunde. [The inheritance of coat colour in the Dachshund.] *Biol. Med.* [*Kbh.*], *4* (6): 1-72.

APGAR, C. S. 1940. The anatomical basis for certain postpartum reactions of the bitch toward her pups in various types of dogs. *Anat. Rec.*, *76* (Suppl. No. 2): 61. (Amer. Ass. Anat.)

BARNES, M. H., and BARNES, M. D. 1946. Colour breeding in Cockers. The evolution of the white. *Tonbridge Free Press Ltd.*, *Kent*, *Engl.* 15 pp.

BARRINGTON, A., LEE, A., and PEARSON, K. 1904. On inheritance of coat-colour in the Greyhound. *Biometrika*, *3*: 245-298.

BARROWS, W. M., and PHILLIPS, J. M. 1915. Colour in Cocker Spaniels. *J. Hered.*, *6*: 387-397.

BAUMANN, F., and HUBER, W. 1946. Über ausgewachsene und juvenile Schädelformen bei verschiedenen Hunderassen. [Adult and juvenile skull forms in different breeds of dog.] *Arch. Klaus-Stift. VererbForsch.*, *21*: 352-361. (Schweiz. Ges. VererbForsch.) [*A.B.A.*, *16*, No. 659.]

BAYER, O. 1936. Massnahmen gegen die Verbreitung schlechter Erbeigenschaften und Degeneration in der Hundezucht. [Measures for the prevention of the spread of deleterious hereditary characters and of degeneration in dog breeding.] *Prag. tierärztl. Arch.*, *16*: 229-238. [*A.B.A.*, *6*: 35.]

BERG, W. 1912. Über stummelschwänzige Katzen und Hunde. Ein Beitrag zu der Lehre von der Reduktion des Schwanzes. [Stump-tailed cats and dogs. A contribution to knowledge on tail reduction.] *Z. Morph. Anthr.*, Sonderheft II: 227-267.

BLEULER, E. 1934. Vererbung erworbener psychischer Eigenschaften. [Inheritance of acquired psychical characters.] *Wien. med. Wschr.*, *84*: 705-707, 740-743.

BOSI, T. 1948. L'impiego della consanguineità nella creazione di una bella famiglia di Boxer. [The use of inbreeding in the creation of a good Boxer family.] *Riv. Zootec.*, *21*: 94-97. [*A.B.A.*, *16*, No. 1544.]

BRIGGS, L. C. 1940. Some experimental matings of color-bred white Bull Terriers. *J. Hered.*, *31*: 236-238. [*A.B.A.*, *8*: 278.]

—— and KALISS, N. 1942. Coat color inheritance in Bull Terriers. *J. Hered.*, *33*: 222-228. [*A.B.A.*, *10*: 256.]

BRINKHOUS, K. M., and GRAHAM, J. B. 1950. Haemophilia in the female dog. *Science*, 111: 723-724. [*A.B.A.*, No. 1548.]

BROUWERS, J. 1950. Goître et hérédité chez le chien. [Goitre and heredity in the dog.] *Ann. Méd. vét.*, *94*: 173-174. [*A.B.A.*, *18*, No. 1102.]

BURNS, M. 1943. Hair pigmentation and the genetics of colour in Greyhounds. *Proc. roy. Soc. Edinb.*, *B*, *61*: 462-490. [*A.B.A.*, *11*: 251.]

CAVAZZA, F. 1931. Un caso di anormale dissociazione di caratteri in un incrocio di razza fra cani. [A case of abnormal segregation in a cross between two breeds of dogs.] *Boll. Zool.*, *2*: 101-110.

COOMBS, R. D. 1917. Bull-terrier breeding. *J. Hered.*, *8*: 314-318.

CREW, F. A. E. 1925. Animal genetics. An introduction to the science of animal breeding. *Edinburgh: Oliver and Boyd.* xvii+420 pp.

DAHL, L. E., and QUELPRUD, T. 1937. Die Vererbung der Haarfarbe beim deutscher Boxer. [The inheritance of coat colour in the German Boxer.] *Z. Zücht.*, *B*, *37*: 159-177. [*A.B.A.*, *5*: 424.]

DAHR, P. 1937. Unterscheidung der Blutgruppen A_1 und A_2 mit α_2 (Anti-o)-Agglutinin enthaltenden Hundeseren. [Differentiation of the blood groups A_1 and A_2 by dog sera containing α_2 (Anti-o)-Agglutinin.] *Klin. Wschr.*, *16*: 204.

DARLING, F. F. 1932. Colour inheritance in Bull Terriers. *The coloured and colour breeding. By T. W. Hogarth. Chapter IV. Galashiels: A. Walker and Son, Ltd.* Pp. 49-50.

—— and GARDNER, P. 1933. A note on the inheritance of the Brindle character in the coloration of Irish Wolfhounds. *J. Genet.*, *27*: 377-378. [*A.B.A.*, *1*: 195.]

DECHAMBRE, P. 1932. La transmission héréditaire des caractères 'liés au sexe'. [Hereditary transmission of sex-linked characters.] *Rev. Zootech.*, *12* (12): 363-370.

DE VILMORIN, P. 1913. Sur les caractères héréditaires des chiens anoures et brachyures. [Inheritance of taillessness and brachyury in the dog.] *C. R. Acad. Sci.* [*Paris*], *157*: 1086-1089.

Dexler, —. 1923. Über chronische konstitutionelle Hydrocephalie beim Hunde. [Chronic constitutional hydrocephalus in the dog.]. *Prag. tierärztl. Arch.*, 1923.

De Zulueta, A. 1949. The hairless dogs of Madrid. *Proc.* 8th *int. Congr. Genet.* 1948 [*Stockh.*]. *Lund*: *Issued as a supplementary volume* of '*Hereditas*', 1949. Pp. 687-688. [Abstract only.] [*A.B.A.*, *17*, No. 1493.]

Dice, L. R. 1942. A family of dog-coyote hybrids. *J. Mammal.*, *23*: 186-192. [*A.B.A.*, *11*: 53.]

Dighton, A. 1923. Coat colour in Greyhounds. *Proc. zool. Soc. Lond.*, 1923 (I): 1-9.

Dressler, W. 1944. Erfahrungen und Erlebnisse bei einer Hundeersatzstaffel. [Experience with a troop of dogs being trained as replacements.] *Der Hund im Kriege. Bd. 1. Erfahrungen über Abrichtung und Einsatz.* [*The dog in war. Vol.* 1. *Experiences in training and use.*] *Z. Hundeforsch.*, N.F., *18*: 28-46.

Druckseis, H. 1935. Geschlechtsverhältnis und Wurfgrösse beim Hund. [Sex ratio and litter size in the dog.] *Vet.-med. Dissertation, Univ. Munich.* 32 pp. [*A.B.A.*, *6*: 128.]

Duke, K. L. 1946. Monozygotic twins in the dog. *Anat. Rec.*, *94*: 35-41. [*A.B.A.*, *14*: 258.]

Englert, H. K. 1938. Ueber die Vererbung der Haarfarben beim Hund. [Inheritance of coat colour in the dog.] *Kleintier u. Pelztier*, *14* (2): [4]+1-38. [*Z. Hundeforsch.*, N.F., *9*.] [*A.B.A.*, *9*:241.]

Esskuchen, E. 1929. Die Färbung der Haussäugetiere in ihrer Entstehung und ihrer Bedeutung für die Widerstandsfähigkeit und ihrer Leistungsfähigkeit derselben. [The origin of colouring in domestic mammals and its significance in their performance and resistance capacity.] *Arb. dtsch. Ges. Züchtungsk.*, H. 42: iv+123 pp.

Ewart, J. Cossar. 1926. Coat of the Lapland wolf. *Proc. roy. Soc. Edinb.*, *46*: 395.

Falaschini, A. 1941. Sul comportamento ereditario dello sperone in '*Canis familiaris*'. [On the mode of inheritance of the dew-claw in the domestic dog.] *Nuova Vet.*, *19*: 126-129. [*A.B.A.*, *12*: 155.]

Freak, M. J. 1948. The whelping bitch. *Vet. Rec.*, *60*: 295-299. Discussion: 300-301, 306. [*A.B.A.*, *16*, No. 1107.]

Frey, M. 1933. Morphologische und histologische Untersuchungen an der Schilddrüse verschiedener Hunderassen. (Ein Versuch, die Konstitution und den Charakter abgewandelter Rassen (Zwergrassen) mit dem Bau und der Funktion der Schilddrüse in Verbindung zu bringen.) [Morphological and histological studies on thyroids of different races of dogs. (Relation between the constitution and character of dwarf races and the structure and function of the thyroid).] *Dissertation, Bonn-Poppelsdorf.* 54 pp.

—— 1934. Morphologische und histologische Untersuchungen an der Hypophyse und Schilddrüse verschiedener Hunderassen in Beziehung auf die einzelnen Konstitutionstypen. [Morphological and histological studies on the pituitary and thyroid of different breeds of dogs in relation to the various types of constitution.] *Endokrinologie, 14*: 116-128. [*A.B.A., 3*: 412.]

FYFE, J. 1950. Lysenko is right. *London*: *Lawrence and Wishart.* 65 pp.

GAIR, R. 1928. Die Wuchsformen des Haarkleides bei Haustieren nach Untersuchungen beim Hunde. [Growth forms of the coat in livestock after investigations on the dog.] *Z. Tierz. ZüchtBiol., 11*: 57-88.

GALTON, F. 1897. The average contribution of each of several ancestors to the total heritages of the offspring. *Proc. roy. Soc., 61*: 401-413.

GASPAR, J. 1930. Analyse der Erbfaktoren des Schädels bei einer Paarung von Ceylon-Nackthund × Dackel. (Fauna et Anatomia ceylanica, IV, Nr. 10.) [Analysis of factors governing skull structure in some crosses of the Ceylon hairless dog and the 'Dackel'.] *Jena. Z. Naturw., 65*: 245-274.

GATES, R. R. 1909. A litter of hybrid dogs. *Science, 29*: 744-747.

GEORGI, W. 1938. Rassen- und funktionelle Merkmale am Unterkiefer des Hundes. [Racial and functional characters of the lower jaw of the dog.] *Kleintier u. Pelztier, 14* (3): 1-55. [*Z. Hundeforsch.*, N.F., *10*.] [*A.B.A., 7*: 240.]

GIRAUD, P., and CIAUDO, P. 1934. Absence de transmission héréditaire des caractères sérologiques de la leishmaniose interne chez le chien. [Absence of hereditary transmission of serological characters in a case of internal Leishmaniosis in the dog.] *C.R. Soc. Biol. Paris, 116*: 433-435. [*A.B.A., 2*: 237.]

GOSSWEILER, R. 1930. Gibt es eine Vererbung von Stummelruten beim Hunden? [Is docked condition of the tail heritable?] *Bull. Soc. fribourg. Sci. nat.*, 1930: 1-14.

GRAHAM, J. B., BUCKWALTER, J. A., HARTLEY, L. J., and BRINKHOUS, K. M. 1949. Canine hemophilia. Observations on the course, the clotting anomaly, and the effect of blood transfusions. *J. exp. Med., 90*: 97-111.

GRAY, H. 1930. Hereditary defects of the eye in dogs. *Vet. Rec., 10*: 1031-1032.

Gray, H. 1933. Congenital deafness in dogs. *Our Dogs*, *91*: 490. [*A.B.A.*, *2*: 237.]

Green, L. G. 1939. South Africa's famous police dogs. *Field*, *174*: 932. [*A.B.A.*, *8*: 66.]

Grüneberg, H., and Lea, A. J. 1940. An inherited jaw anomaly in long-haired Dachshunds. *J. Genet.*, *39*: 285-296. [*A.B.A.*, *8*: 160.]

Guerreiro, R. T. [da] C. 1947. Variações digitais do pé do cão. [Digital variations of the foot in the dog.] *Rev. Med. vet.* [*Lisboa*], *42*: 43-85. [*A.B.A.*, *16*, No. 1546.]

Hachlov, V. 1930. Über die Genetic der Hunde. [Genetics of the dog.] *Züchter*, *2*: 261-263.

Hagedoorn, A. L. 1912. On tricolour coat in dogs and guinea-pigs. *Amer. Nat.*, *46*: 682-683.

Haldane, J. B. S. 1927. The comparative genetics of colour in rodents and carnivora. *Biol. Rev.*, *2*: 199-212.

Hall, R. E. 1943. Cranial characters of a dog-coyote hybrid. *Amer. Midl. Nat.*, *29*: 371-374. [*A.B.A.*, *12*: 39.]

Hammond, J. 1932. Growth and the development of mutton qualities in the sheep. A survey of the problems involved in meat production. *Edinburgh*: *Oliver and Boyd*. xxvi + 597 pp.

Hancock, J. L., and Rowlands, I. W. 1949. The physiology of reproduction in the dog. *Vet. Rec.*, *61*: 771-776. Discussion: 777-779. [*A.B.A.*, *18*, No. 304.]

Hare, T. 1932. A congenital abnormality of hair follicles in dogs resembling trichostasis spinulosa. *J. Path. Bact.*, *35*: 569-571.

Härtl, J. 1938. Die Vererbung des Kryptorchismus beim Hund. [The inheritance of cryptorchidism in dogs.] *Kleintier u. Pelztier*, *14* (1): iv + 1-37 pp. [*Z. Hundeforsch.*, N.F., *8*.] [*A.B.A.*, *8*: 278.]

Hauck, E. 1941. Wie kommt der Winkelkopf zustande? [How does frog-face (down-face) occur?] *Wien. tierärztl. Mschr.*, *28*: 443-447. [*A.B.A.*, *11*: 188.]

Haynes, W. 1913. Prepotency in Airedale Terriers. *Science*, *38*: 404-405.

—— 1914. Inbreeding in dogs. Statistical study of the pedigrees of two typical breeds. *J. Hered.*, *5*: 368-369.

Heape, W. 1908. Notes on the proportions of the sexes in dogs. *Proc. Camb. phil. Soc.*, *14*: 121-151.

Hermann, G. R. 1926. The heart of the racing Greyhound. *Proc. Soc. exp. Biol.* [*N.Y.*], *23*: 856.

Hippen, E. v. 1930. Embryologische Untersuchungen über Vererbung angeborener Katarakt, über Schichtstar des Hundes sowie über eine besondere Form von Kapselkatarakt. [Embryological investigations on the inheritance of congenital cataract, lamellar cataract in the dog and a particular form of capsular cataract.] *Graefes Arch.*, *124*: 300-324.

Hirschfeld, W. K. 1933. Haarfarbenvererbung bei hochbeinigen Terriern. [Colour inheritance in 'Long-legged' Terriers.] *Züchter*, *5*: 141-144. [*A.B.A.*, *1*: 279.]

—— 1937. Genetische Untersuchungen über die Haarfarbe beim Hunde. [Genetical investigations on hair colour in dogs.] *Dissertation, Giessen.*

Hodgman, S. F. J., Parry, H. B., Rasbridge, W. J., and Steel, J. D. 1949. Progressive retinal atrophy in dogs. 1. The disease in Irish Setters (Red). *Vet. Rec.*, *61*: 185-189. [*A.B.A.*, *17*, No. 644.]

Hogarth, T. W. 1933. The seven sources of colour in Bull Terriers. *Our Dogs*, *93*: 1011-1012. [*A.B.A.*, *2*: 238.]

Horlacher, L. J. 1927. Sheep production. *New York and London*: *McGraw-Hill Book Co.* x+418 pp.

Høst, P., and Sreinson, S. 1936. [Hereditary cataract in the dog.] *Norsk Vet.-Tidsskr.*, *48*: 244-270.

Huber, W. 1948. Die Beurteilung der Hundeschnauze als genetisches Problem. [The evaluation of the muzzle of the dog as a genetical problem.] *Arch. Klaus-Stift. VererbForsch.*, *23*: 486-492. (Schweiz. Ges. VererbForsch.) [*A.B.A.*, *17*, No. 1069.]

—— 1948. Die Gaumenspalte beim Pekingesen. [Cleft palate in the Pekingese.] *Arch. Klaus-Stift. VererbForsch.*, *23*: 492-496. (Schweiz. Ges. VererbForsch.) [*A.B.A.*, *17*, No. 1070.]

Hudson, P. S., and Richens, R. H. 1946. The new genetics in the Soviet Union. *Cambridge*: *Imperial* [now *Commonwealth*] *Bureau of Plant Breeding and Genetics.* 88 pp. [French, Spanish, German and Russian summaries.] [*A.B.A.*, *14*: 187.]

Humphrey, E. S. 1934. 'Mental tests' for Shepherd Dogs. *J. Hered.*, *25*: 128-136. [*A.B.A.*, *2*: 239.]

Humphrey, E. [S.], and Warner, L. 1934. Working dogs; an attempt to produce a strain of German Shepherds which combines working ability and beauty of conformation. *Baltimore*: *Johns Hopkins Press.* xiv+253 pp.

Hutt, F. B., Rickard, C. G., and Field, R. A. 1948. Sex-linked hemophilia in dogs. *J. Hered.*, *39*: 2-9. [*A.B.A.*, *16*, No. 1105.]

Huxley, J. 1949. Soviet genetics and world science. Lysenko and the meaning of heredity. *London*: *Chatto and Windus.* x+245 pp. [*A.B.A.*, *17*, No. 1655.]

Ibsen, H. L. 1916. Tri-colour inheritance. *Genetics*, *1*: 367-376.

Ilchester, Earl of. 1941. A history of Yellow Retrievers. *Field*, *178*: 496. [*A.B.A.*, *10*: 50.]

ILJIN, N. A. 1926. [Principal laws of inheritance and kynology.] *Dog Breeding and Training U.S.S.R.*, No. 1.

—— 1926. Rubinovoglazie životnyh i ego nasledovanie. II. Rubinovoglazie sredi mlekopitajuščih. [Ruby eye in animals and its inheritance. II. Ruby eye in mammals.] *True. Lab. èksp. Biol.* [*Mosk.*], *1*: 121-127, 129. [English summary.]

—— 1928. [Ruby eye in dogs.] *Dog Breeding and Training U.S.S.R.*, No. 1 (18): 1-3.

—— 1931. O nasledovanii okraski u Dobermann-Pinčera. [Coat colour inheritance in Dobermann-Pinscher.] *Trud. Dinam. Razvit.*, *6*: 241-251. [English summary.]

—— 1931. Über die Vererbung der Färbung beim Dobermann-Pinscher. [Inheritance of colour in the 'Dobermann-Pinscher.'] *Züchter*, *3*: 370-376.

—— 1932. Genetika i razvedenie sobak. [Genetics and the breeding of dogs.] *Moscow-Leningrad*: *Seljskohozgiz.* 164 pp. [*A.B.A.*, *1*: 61.]

—— 1934. Rasščeplenie pri skreščivanii volka i sobaki i materialy po genetike domašneĭ sobaki. [Segregation in a cross between a wolf and a dog and material on the genetics of the dog.] *Trud. Dinam. Razvit.*, *8*: 105-166. [English summary.]

—— 1937. La variabilité parallèle dans la phénogenèse de la phénogenèse de la conque de l'oreille chez les canides. [Parallel variability in the phenogenesis of the concha in the ear of Canidae.] *C.R.* (*Dokl.*) *Acad. Sci. U.R.S.S.*, N.S., *17*: 379-383. [*A.B.A.*, *6*: 311.]

—— 1941. Wolf-dog genetics. *J. Genet.*, *42*: 359-414. [*A.B.A.*, *10*: 51.]

JÄKEL, H. 1940. Mikroskopische Untersuchungen über die Epidermalgebilde an verschiedenen Körperstellen mehrerer Hunderassen. [Microscopic investigations on the epidermal structure on various body parts in several breeds of dog.] *Dissertation, Vienna.*

JAMES, W. T. 1951. Social organization among dogs of different temperaments, terriers and beagles, reared together. *J. comp. physiol. Psychol.*, *44*: 71-77.

JOURNAL OF HEREDITY. 1917. The hairless dog. *J. Hered.*, *8*: 519-520.

KEELER, C. E. 1940. The inheritance of predisposition to renal calculi in the Dalmatian. *J. Amer. vet. med. Ass.*, *96*: 507-510. [*A.B.A.*, *9*: 48.]

—— 1940. The Dalmatian psyche. *J. Hered.*, *31*: 112. [*A.B.A.*, *8*: 279.]

—— and TRIMBLE, H. C. 1938. The inheritance of dew claws in the dog. *J. Hered.*, *29*: 145-148. [*A.B.A.*, *6*: 129.]

KEELER, C. E. and TRIMBLE, H. C. 1940. Inheritance of position preference in coach dogs. *J. Hered.*, *31*: 50-54. [*A.B.A.*, *8*: 279.]

KELLEY, R. B. 1947. Sheep dogs. Their breeding, maintenance and training. *Sydney and London*: *Angus and Robertson*. 2nd ed. of 'Animal Breeding'. x+210 pp. [*A.B.A.*, *16*, No. 795.]

KITTEL, H. 1931. Ueber Dermoide der Kornea und Spaltbildungen der Lider am Auge von Bernhardinerhunden. [Dermoid cysts of the cornea and fissures of the eyelids in the St. Bernard dogs.] *Dtsch. tierärztl. Wschr.*, 1931 (52): 793-797.

KLARENBEEK, A., KOOPMANS, S., and WINSSER, J. 1942. Een aanvalsgewijs optredende stoornis in de regulatie van de spiertonus, waargenomen bij schotsche terriers. [Muscular disturbances in Scottish Terriers.] *Tijdschr. Diergeneesk.*, *69*: 14-21. [German, English and French summaries.] [*A.B.A.*, *12*: 214.]

KLATT, B. 1933. Gefangenschaftsveränderungen bei Säugetieren und ihre Bedeutung für die Fragen der Domestikation. [Changes in mammals during captivity and their significance in domestication.] *Nova Acta Leop. Carol.*, *1*: 325-326. [*A.B.A.*, *2*: 298.]

—— 1939. Erbliche Missbildungen der Wirbesäule beim Hund. [Heritable anomalies of the vertebral column of the dog.] *Zool. Anz.*, *128*: 225-235. [*A.B.A.*, *9*: 241.]

—— 1941. Kreuzungen an extremen Rassetypen des Hundes (Bulldogge-Windhundkreuzungen.) [Crosses of extreme breed types of dogs. (Bulldog × Greyhound crosses).] *Z. menschl. Vererb.- u. KonstLehre*, *25*: 28-93. [*A.B.A.*, *11*: 53.]

—— 1942. Kreuzungen an extremen Rassetypen des Hundes. 2. Die Verhältnisse des Kopfes und der Physiognomie im allgemeinen. [Crossing of extreme types of canine breeds. 2. The proportions of head and general physiognomy.] *Z. menschl. Vererb.- u. Konst-Lehre*, *26*: 320-356. [*A.B.A.*, *16*, No. 661.]

—— 1944. Kreuzungen an extremen Rassetypen des Hundes. IV. Teil: Schädel und Physiognomie der Kreuzungstiere. [Crosses between extreme breed types of dog. IV. Skull and physiognomy of the crossbred animals.] *Z. menschl. Vererb.- u. KonstLehre*, *28*: 113-158. [*A.B.A.*, *17*, No. 243.]

—— 1949. Messend-anatomische Untersuchungen an gegensätzlichen Wuchsformtypen. [Investigations pertaining to the measurement and anatomy of contrasting growth form types.] *Roux' Arch. EntwMech. Organ.*, *143*: 573-592. [*A.B.A.*, *17*, No. 1065.]

KLEEMANN, —. 1937. Abstammung und Entwicklung von Deutsch-Kurzhaar. [The origin and development of the German Pointer.] *Kleintier u. Pelztier*, *13* (8): 33 pp. [*Z. Hundeforsch.*, N.F., *6*.]

KLODNITZKY, J., and SPETT, G. 1925. Kurzschwänzige und schwanzlose Variation bei Hunden. [Short-tailed and tailless varieties in the dog.] *Z. indukt. Abstamm.- u. VererbLehre*, *38*: 72-74.

Koch, W. 1935. Neue pathogene Erbfaktoren bei Hunden. [New pathological hereditary characters in the dog.] *Z. indukt. Abstamm. -u. VererbLehre*, *70*: 503-506. (Dtsch. Ges. Vererbungsw.)

—— 1938. Beiträge zur Erbpathologie der Haustiere. I. Kryptorchismus bei Wachtelhunden. [On hereditary defects in domesticated animals. I. Cryptorchism in the Spaniel.] *Tierärztl. Rdsch.*, *44*: 302. [*A.B.A.*, *8*: 405.]

Kohn, F. G. 1911. Beitrag zur Kenntnis der Haut des Nackthundes. [The skin of the hairless dog.] *Zool. Jb.*, *Anat.*, *31*: 427-438.

Kohts, A. E. 1948. The variation of colour in the common wolf and its hybrids with domestic dogs. *Proc. zool. Soc. Lond.*, *117*: 784-790. [*A.B.A.*, *16*, No. 1106.]

Kollarits, J. 1924. Das Dauerzittern mancher Rassenhunde als Heredo-degeneration. [Permanent trembling in some dog breeds as hereditary degeneration.] *Schweiz. med. Wschr.*, *54*: 1131-1132.

Konde, W. N. 1947. Congenital subluxation of the coxofemoral joint in the German Shepherd Dog. *N. Amer. Vet.*, *28*: 595-599. [*A.B.A.*, *17*, No. 244.]

Krušinskiĭ, L. V. 1938. Issledovanie po fenogenetike priznakov povedenija u sobak. [Studies on the phenogenetics of behaviour in dogs.] *Biol. Ž.* [*Mosk.*], *7*: 869-891. [English summary.] [*A.B.A.*, *8*: 161.]

—— 1944. Nasledstvennoe 'fiksirovanie' individualjno priobretennogo povedenija životnyh i proishodženie instinktov. [The hereditary 'fixing' of individually acquired behaviour of animals and the origin of instincts.] *Ž. obšč. Biol.*, *5*: 261-283. [English summary.] [*A.B.A.*, *14*: 104.]

Krushinsky [Krušinskiĭ], L. V. 1946. Correlation between constitution and behaviour in dogs. *C.R.* (*Dokl.*) *Acad. Sci. U.R.S.S.*, N.S., *52*: 633-636. [*A.B.A.*, *17*, No. 240.]

Krušinskiĭ, L. V. 1946. Rolj nasledstvennosti i usloviĭ vospitanija v projavlenii i vyraženii priznakov povedenija u sobak. [On the role of hereditary and non-hereditary factors in the manifestation and expression of symptoms of the dog's behaviour.] *Izv. Akad. Nauk SSSR.*, *Ser. biol.*: 69-81. [English summary.] [*A.B.A.*, *14*: 168.]

—— 1946. Polovoe različie v povedenii sobak. [The sex difference in the behaviour of dogs.] *Ž. obšč. Biol.*, *7*: 131-146. [English summary.] [*A.B.A.*, *16*, No. 660.]

Kurzbauer, W., and Marchlewski, T. 1938. Zagadnienie ewolucji dominacji w hodowli zwierząt domowych. I. Obserwacje nad psem domowym. [The problem of evolution of dominance in breeding domestic animals. I. Observations on the domestic dog.] *Kosmos* [*Lwów*], *A*, *63*: 121-144. [English summary.] [*A.B.A.*, *7*: 142.]

Lacroix, J. V., Park, S. E., and Adler, A. E. (Jr.). 1942. Ascorbic acid blood levels in the dog. *N. Amer. Vet.*, *23*: 329-331. [*A.B.A.*, *12*: 155.]

Lang, —. 1910. Über alternative Vererbung bei Hunden. [Alternative inheritance in dogs.] *Z. indukt. Abstamm.- u. VererbLehre*, *3*: 1-33.

Langner, G. 1935. Welcher Hund der sechs Gebrauchshundrassen ist für den Dienst bei der Reichsbahn der geeignetste? [Which of the six utility breeds of dogs is best adapted for service on the railway?] *Tierärztl. Rdsch.*, *41*: 518-520. [*A.B.A.*, *4*: 339.]

Lauer, A. 1930. Blutgruppendifferenzierung bei Hunden. [Blood group differentiation in dogs.] *Z. ImmunForsch.*, *68*: 433-435.

Lea, A. J. 1943. The inheritance of coat and nose colour in long-haired Dachshunds. *J. Genet.*, *45*: 197-205. [*A.B.A.*, *11*: 252.]

Letard, É. 1930. L'hérédité mendélienne du caractère 'peau nue' chez le chien. [Mendelian inheritance of the character 'hairless' in the dog.] *C.R. Soc. Biol. Paris*, *103*: 1135-1136.

—— 1930. Le Mendélisme expérimental. Expériences sur l'hérédité du caractère 'peau nue' dans l'espèce canine. Observations sur la transmission de quelques autres attributs. [Experimental Mendelism. Experiments on the inheritance of hairlessness in the dog; observations on the inheritance of some other characters.] *Rev. vét.* [*Toulouse*], *82*: 378.

—— 1934. Les applications de la génétique à l'élevage des animaux domestiques. [Application of genetics to the breeding of domestic animals.] *12th int. Vet. Congr.* [*New York*], *3*: 437-499.

Lienhart, R. 1932. Contribution à l'étude de l'hérédité chez les chiens anoures et brachyures. [Inheritance in tailless and short-tailed dogs.] *C.R. Soc. Biol. Paris*, *110*: 1164-1168.

Little, C. C. 1914. Inheritance of coat colour in Pointer dogs. *J. Hered.*, *5*: 244-248.

—— 1934. Inheritance in Toy Griffons. *J. Hered.*, *25*: 198-200.

—— 1948. Genetics in Cocker Spaniels. Observations on heredity and on physiology of reproduction in American Cocker Spaniels. *J. Hered.*, *39*: 181-185. [*A.B.A.*, *16*, No. 1547.]

—— and Jones, E. E. 1919. Coat colour in Great Danes. *J. Hered.*, *10*: 309-320.

Lotsy, J. P. 1923. A peculiar eye-color among Malamute-dogs. *Genetica*, *5*: 77-78.

LUMER, H. 1940. Evolutionary allometry in the skeleton of the domesticated dog. *Amer. Nat., 74*: 439-467. [*A.B.A., 9*: 143.]

LUSH, J. L. 1945. Animal breeding plans. *Ames: Iowa State College Press.* 3rd ed. viii+443 pp.

LUTZ, F. E., and MEEK, E. B. 1902. Inheritance of color among Pointers. *Science, 15*: 571-572.

LYSENKO, T. D. 1951. Heredity and its variability. *Moscow: Foreign Languages Publishing House.* 136 pp.

MCCULLOCH, J. H. 1946. Sheep dogs and their masters. Being a history of the Border Collie, together with some notes about the men who have developed the breed. *Edinburgh and London: Moray Press.* Rev. ed. 120 pp. [*A.B.A., 16*, No. 344.]

—— [1952.] Border Collie studies. *Edinburgh: Pentland Press.* 48 pp. [*A.B.A., 20*, No. 1350.]

MCNELLIS, R. 1944. The Peruvian Bull Terrier: new breed of dog. *J. Amer. vet. med. Ass., 104*: 281-282. [*A.B.A., 12*: 213.]

MAGNUSSON, H. 1911. Über Retinitis pigmentosi und Konsanguinität beim Hunde. [Retinitis pigmentosi and consanguinity in the dog.] *Arch. vergl. Ophthal., 2*: 147-163.

—— 1917. Noch ein Fall von Nachtblindheit beim Hunde. [Another case of night blindness in the dog.] *Arch. Ophthal., 93*: 404-411.

MALONE, J. Y. 1918. Spermatogenesis in the dog. *Trans. Amer. micr. Soc., 37*: 97-100.

MARCHLEWSKI, T. 1926. Craniological studies on dogs. *Bull. int. Acad. Cracovie, B*: 601-614.

—— 1930. Genetic studies on the domestic dog. *Bull. int. Acad. polon. Sci. Lett., Cl. Sci. math. nat., B (II)*: 117.

—— 1930. Craniology of the domestic dog. *Bull. int. Acad. polon. Sci. Lett., Cl. Sci. math. nat., B (II)*: 511-548.

—— 1941. Change of dominance in canine colour genetics. *Proc. 7th int. genet. Congr.* [*Edinb.*]: 211. [*A.B.A., 9*: 278.]

—— 1947. Studies in the evolution of dominance in mammals. *Bull. int. Acad. polon. Sci. Lett., Cl. Sci. math. nat., B (II)*, 1946 (1/10): 107-110.

MERKENS, J. 1938. Haemophilie bij honden. [Haemophilia in the dog.] *Ned.-ind. Bl. Diergeneesk., 50*: 149-151. [*A.B.A., 8*: 406.]

MILLAIS, E. [1895.] Two problems of reproduction. Lecture delivered at St. Thomas' Hospital on February 25th, 1895. *Manchester: 'Our Dogs' Publishing Co., Ltd.* 51 pp.

MINOUCHI, O. 1928. The spermatogenesis of the dog, with special reference to meiosis. *Jap. J. Zool., 1*; 255-268.

MITCHELL, A. L. 1935. Dominant dilution and other color factors in Collie dogs. *J. Hered., 26*: 424-430. [*A.B.A., 4*: 80.]

Moltzen-Nielson, H. 1937. Calvé-Perthes-Krankheit, Malum deformans juvenilis coxae bei Hunden. [Calvé-Perthe disease. A juvenile hip deformity in dogs.] *Arch. Tierheilk.*, *72*: 91.

Morton, A. G. 1951. Soviet genetics. *London*: *Lawrence and Wishart.* 174 pp.

Nachtsheim, H. 1933. Tierzüchterische Ergebnisse des VI. internationalen Kongresses für Vererbungswissenschaft, Ithaca, 1932. [Conclusions of the 6th International Congress on Genetics and Animal Breeding, Ithaca, 1932.] *Züchtungskunde*, *8*: 193-213. [*A.B.A.*, *1*: 154.]

—— 1949. Vom Wildtier zum Haustier. [From the wild to the domestic animal.] *Berlin and Hamburg*: *Paul Parey.* 2nd ed. 123 pp.

Obouissier, H. 1940. Über den Einfluss der Domestikation auf die Hypophyse. [The influence of domestication on the hypophysis.] *Zool. Anz.*, *132*: [26 pp.] [*A.B.A.*, *13*: 49.]

—— 1942. Das Verhalten der Hypophyse bei Kreuzungen extremer Rassetypen des Hundes. [Behaviour of the hypophysis in the crossing of extreme types of canine breeds.] *Z. menschl. Vererb.-u. KonstLehre*, *25*: 663-694. [*A.B.A.*, *16*, No. 663.]

Oguma, K., and Kakino, S. 1932. A revised check list of the chromosome number in Vertebrata. *J. Genet.*, *26*: 239-254.

Ollerhead, K. 1939. An unusual condition met with in English Setters. *Vet. Rec.*, *51*: 200-201. [*A.B.A.*, *7*: 142.]

Olson, W. H. 1940. Isoagglutinins in the blood plasma of dogs. *Amer. J. Physiol.*, *129*: 433-434. (Proc. Amer. physiol. Soc.) [*A.B.A.*, *8*: 434.]

Onslow, H. 1923. The relation between uric acid and allantoin excretion in hybrids of the Dalmatian hound. *Bio-chem. J.*, *17*: 334-340.

—— 1923. Uric acid and allantoin excretion among offspring of Dalmatian hybrids. *Bio-chem. J.*, *17*: 564-568.

Painter, T. S. 1925. A comparative study of the chromosomes of mammals. *Amer. Nat.*, *59*: 385-409.

Pardoe, J. H. 1949. Fox Terriers. *London: Williams and Norgate, Ltd.* 119 pp. [*A.B.A.*, *18*, No. 431.]

Parry, H. B. 1950. Viral hepatitis of dogs (Rubarth's disease). *Vet. Rec.*, *62*: 559-565.

Pearson, K., and Usher, C. H. 1929. Albinism in dogs. *Biometrika*, *21*: 144-163.

Peters, H. B. 1938. Ergänzendes zur Kenntnis der Chincha-Bulldogge. [Further data on the Chincha Bulldog.] *Z. Hundeforsch.*, N.F., *13*: 49-54. [*A.B.A.*, *8*: 280.]

Peters, H. B. 1938. Der Karakatschan-Hund. [The Karakatchan dog.] *Z. Hundeforsch.*, N.F., *13*: 55-62. [*A.B.A.*, *8*: 280.]

Phillips, J. M. 1937. Albinism in a Cocker Spaniel. *J. Hered.*, *28*: 103-104. [*A.B.A.*, *5*: 176.]

—— 1938. Sable coat colour in Cockers. *J. Hered.*, *29*: 67-69. [*A.B.A.* *6*: 130.]

—— 1945. 'Pig Jaw' in Cocker Spaniels. Retrognathia of the mandible in the Cocker Spaniel and its relationship to other deformities of the jaw. *J. Hered.*, *36*: 177-181. [*A.B.A.*, *13*: 161.]

—— and Felton, T. M. 1939. Hereditary umbilical hernia in dogs. *J. Hered.*, *30*: 433-435. [*A.B.A.*, *8*: 67.]

—— and Knight, E. D. 1938. Merle or Calico Foxhounds. *J. Hered.*, *29*: 365-367. [*A.B.A.*, *6*: 312.]

Pickett, F. N. 1950. The book of the Alsatian dog. *London*: *The Weald Press, Ltd.* 176 pp.

Pocock, R. I. 1907. On the black and tan pattern in domestic dogs. *Ann. Mag. nat. Hist.* [7th Ser.], *19*: 192-194.

Prinzhorn, F. 1921. Die Haut und die Rückbildung der Haare beim Nackthunde. [The skin and involution of the hair in hairless dogs.] *Jena. Z. Naturw.*, *57*: 143-198.

Pullig, T. 1950. Inheritance of whorls in Cocker Spaniels. A preliminary report. *J. Hered.*, *41*: 239-242. [*A.B.A.*, *19*, No. 825.]

Raghavachari, K. 1943. Some observations on rickets in pups. *Indian J. vet. Sci.*, *13*: 137-140. [*A.B.A.*, *12*: 155.]

Rcheulishvili [Rčeusvili], M. 1938. Shorttailedness (brachyury) in Georgian dogs and its inheritance. *C. R.* (*Dokl.*) *Acad. Sci. U.R.S.S.*, N.S., *19*: 535-538. [*A.B.A.*, *7*: 142.]

Richter, Johannes. 1931. Züchterische Versuche über die Erblichkeit des Kropfes bei Hunden. [Breeding experiments on the inheritance of goitre in dogs.] *Z. Zücht.*, *B*, *21*: 483-503.

Ritter, R. 1933. Anomalien an Hundegebissen. [Dental anomalies in dogs.] *Fortschr. Orthod.*, *3* (1): 10-20.

—— [1937.] Können Anomalien des Gebisses gezüchtet werden? [Can teeth anomalies be produced by breeding?] *Dtsch. Zahn-Mund- u. Kieferheilk.*, *4*: 235-257. [*A.B.A.*, *6*: 223.]

Robinson, K., and Lee, D. H. K. 1941. Reactions of the dog to hot atmospheres. *Proc. roy. Soc. Qd*, *53*: 177-188.

Scott, J. P., and Fuller, J. L. 1951. Research on genetics and social behavior at the Roscoe B. Jackson Memorial Laboratory, 1946-1951—a progress report. *J. Hered.*, *42*: 191-197. [*A.B.A.*, *20*, No. 387.]

Seiferle, E. 1927. Atavismus und Polydaktylie der hyperdaktylen Hinterpfoten des Haushundes. [Atavism and polydactyly of the hyperdactyl hindpaws of the dog.] *Gegenbaurs Jb.*, *57*: 313-380.

—— 1928. Wesen, Verbreitung und Vererbung hyperdaktyler Hinterpfoten beim Haushund. [Nature, frequency and inheritance of polydactyly in the hind-feet of the dog.] *Schweiz. Arch. Tierheilk.*, *70*: 354-377, 415-437.

SERPUKHOVITIN, D. V. 1936. [Topographical history of the skin of the dog.] *Sci. Mem. Univ. Perm*, *2* (4): 51-69. [Russian with English summary.]

SMITH, A. Croxton. 1939. Bloodhound-Basset crosses. *Field*, *174*: 896. [*A.B.A.*, *8*: 67.]

—— 1939. Crossbred dogs. *Field*, *174*: 939. [*A.B.A.*, *8*: 67.]

—— 1941. Dogs suitable for the Army. The German experience. War dogs at work. *Field*, *178*: 334; 540. [*A.B.A.*, *10*: 51.]

SMYTHE, R. H. 1945. Recurrent tetany in the dog. *Vet. Rec.*, *57*: 380. [*A.B.A.*, *13*: 221.]

SOMMER, O. 1931. Untersuchungen über die Wachstumsvorgänge am Hundeskelett. [Investigations on the skeletal growth processes in the dog.] *Arch. Tierernähr. Tierz.*, *6*: 439-469.

SPREULL, J. S. A. 1949. Some observations on breeding, whelping and rearing. *Vet. Rec.*, *61*: 579-582. [*A.B.A.*, *17*, No. 1499.]

STEIGER, A. L. v. 1936. Über die Haar-Grundfarben beim Jagdhund und ihre genetischen Beziehungen zum Nasen- und Irispigment. [The basic coat colours in gun dogs and their genetic relation to nose and iris pigmentation.] *Züchter*, *8*: 261-271. [*A.B.A.*, *6*: 38.]

—— 1938. Über die Vererbung der Haarfarben beim Hund. [On inheritance of coat colour in the dog.] *Z. Hundeforsch.*, N.F., *13*: 79-83. [*A.B.A.*, *8*: 280.]

STEINIGER, F. 1940. Die Vererbung des Schwanzverlustes. [The inheritance of taillessness.] *Ztg Fachsch. dtsch. Schäferhunde*, Nr. 23/24. [*A.B.A.*, *11*: 252.]

—— 1940-42. Die erbliche Hasenscharte des Hundes. [Hereditary hare-lip in the dog.] *Arb. ReichsgesundhAmt.*, *74*: 399-404. [*A.B.A.*, *13*:221.]

STOCKARD, C. R. 1928. Inheritance of localised dwarfism and achondroplasia in dogs. *Anat. Rec.*, *38*: 29.

—— 1936. An hereditary lethal for localized motor and preganglionic neurones with a resulting paralysis in the dog. *Amer. J. Anat.*, *59*: 1-53. [*A.B.A.*, *5*: 177.]

—— and collaborators. 1941. The genetic and endocrinic basis for differences in form and behavior as elucidated by studies of contrasted pure-line dog breeds and their hybrids. With special contributions on behavior by O. D. Anderson and W. T. James.

Amer. anat. Mem., No. 19. *Philadelphia: Wistar Institute of Anatomy and Biology*. xx+775 pp. [*A.B.A.*, *11*: 138.]

Taylor, G. F. [No date.] Hereditary blindness in Irish Setters. *Personal communication.* [1 p.] [Typescript.]

Thorne, F. C. 1944. The inheritance of shyness in dogs. *J. genet. Psychol.*, *65*: 275-279. [*A.B.A.*, *14*: 257.]

Tichota, J. 1937. Das Verwandtschaftsverhältnis des australischen Dingos zu den prähistorischen Typen des Haushundes—Kraniologische Studie. [The relationship between the dingo and prehistoric types of the domestic dog: a craniological study.] *Zool. Anz.*, *120*: 177-190. [*A.B.A.*, *6*: 130.]

Tjebbes, K., and Wriedt, C. 1927. The albino factor in the Samojede dog. *Hereditas* [*Lund*], *10*: 165-168.

Trimble, H. C., and Keeler, C. E. 1938. The inheritance of 'high uric acid excretion' in dogs. *J. Hered.*, *29*: 280-289. [*A.B.A.*, *7*: 38.]

—— —— 1939. Preference of Dalmatian dogs for particular positions in coach running, and inheritance of this character. *Nature* [*Lond.*], *144*: 671-672. [*A.B.A.*, *7*: 341.]

Tudor-Williams, V. 1946. The barkless dogs of Central Africa. *J. Soc. Pres. Fauna Emp.*, Pt. 54: 31-33. [*A.B.A.*, *18*, Nò. 711.]

Uray, H. 1937. Vererbte Stummelschwänze beim Hunde. [Inherited stumpy tail in the dog.] *Tierärztl. Rdsch.*, *43*: 633. [*A.B.A.*, *7*: 242.]

Ušakova, M. T. 1946. K voprosy o vozrastnoĭ izmenčivosti sobak različnyh porod. [On the question of age variability in dogs of different breeds.] *Dokl. nauč. Konf. Timirjazev seljskohoz. Akad.*, 1946 (3): 169-171. [*A.B.A.*, *14*: 169.]

Vau, E. 1939. Die Wanderung des knöchernen äusseren Gehörganges als Rassemerkmal. (Untersuchungen an Rind und Hund.) [The course of the bony passage of the outer ear as a breed character. Researches on cattle and dogs.] *Z. Anat. EntwGesch.*, *109*: 161-181. [*A.B.A.*, *8*: 100.]

Verlinde, J. D., and Ojemann, J. G. 1946. Eeine aangeboren misvormingen van het centrale zenuwstelsel. [Some congenital abnormalities of the central nervous system.] *Tijdschr. Diergeneesk.*, *71*: 557-564. [English summary.]

Veterinary Record. 1930. Defects in dogs. Malformation and heredity. *Vet. Rec.*, *10*: 933.

Vicari, E. M. 1932. Inheritance of thyroid-size and thyroid-structure in six crosses of pure-bred dogs. *Proc. 6th int. Congr. Genet.* [*Ithaca, N.Y.*], *2*: 200-201.

VICARI, E. M. 1934. Ulna-radius index associated with chondrodystrophy in dogs. *Amer. Nat.*, *68*: 171-172. (Genet. Soc. Amer.)

VOGEL, H. H. (Jr.). 1947. Social relations among Springer Spaniel dogs. *Anat. Rec.*, *99*: 625.

WAGNER, K. 1930. Rezente Hunderassen. Eine osteologische Untersuchung. [Modern races of dogs—an osteological investigation.] *Skr. norske VidenskAkad.*, *Mat.-naturv. Kl.*, 1929 (9): 157 pp.

WARREN, D. C. 1927. Coat colour inheritance in Greyhounds. *J. Hered.*, *18*: 513-522.

WEATHERFORD, H. L., and TRIMBLE, H. C. 1940. A further morphological and biochemical study on the intranuclear crystals in the hepatic cells of the dog—the pure breed and hybrid Dalmatian. *Anat. Rec.*, *77*: 487-507. [*A.B.A.*, *9*: 49.]

WEISS, M. 1940. The Dalmatian psyche. *J. Hered.*, *31*: 112. [*A.B.A.*, *8*: 280.]

WELLMANN, O. 1916. Kreuzungsversuche mit kurzhaarigen, schwarzbraunen Dachshund und kurzhaarigen Foxterrier. [A crossing experiment with a short-haired, black-brown Dachshund and a short-haired Fox Terrier.] *Állattani Közlemínyek. Budapest.* Pp. 248-262.

WERTH, E. 1944. Die primitiven Hunde und die Abstammungsfrage des Haushundes. [Primitive dogs and the question of the descent of domestic dogs.] *Z. Tierz. ZüchtBiol.*, *56*: 213-260. [*A.B.A.*, *14*: 41.]

WHITNEY, L. F. 1927. The mating cycle of the dog. *Chase Mag.*

—— 1928. The inheritance of a ticking factor in hounds. *J. Hered.*, *19*: 498-502.

—— 1929. Heredity of the trail barking propensity in dogs. *J. Hered.*, *20*: 561-562.

—— 1932. Inheritance of mental aptitudes in dogs. *Proc. 6th int. Congr. Genet.* [*Ithaca*, *N.Y.*], *2*: 211-212. [*A.B.A.*, *1*: 42.]

—— 1934. Colour inheritance in the Doberman Pinscher. *Dog News*, Aug. and Sep., 1934.

—— 1939. The sex ratio of dogs maintained under similar conditions. *J. Hered.*, *30*: 388-389. [*A.B.A.*, *8*: 67.]

—— 1946. The successful transfer of ovaries between dogs of different breed. *Science*, *103*: 654-655. [*A.B.A.*, *14*: 169.]

—— 1947. Ovarian transplantation in dogs. *Vet. Med.*, *42*: 30-32. [*A.B.A.*, *17*, No. 648.]

—— 1948. How to breed dogs. *New York*: *Orange Judd Publishing Co.*, *Inc.* Rev. ed. xviii+418 pp. [*A.B.A.*, *17*, No. 342.]

WINGE, Ö. 1920. Über die Vererbung der Haarfarbe der Pferde.

[The inheritance of coat colour in the horse.] *Z. indukt. Abstamm.-u. VererbLehre*, *24*: 1-32.

—— 1946. On the bicolour gene in dog. *C. R. Lab. Carlsberg, Sér. physiol.*, *24*: 125-132. [*A.B.A.*, *14*: 257.]

—— 1950. Inheritance in dogs with special reference to hunting breeds. Translated by C. Roberts. *New York: Comstock Publishing Co.* ix+153 pp. [*A.B.A.*, *19*, No. 432.]

WINZENBURGER, W. 1936. Die Häufigkeitsverteilung der Geschlechtskombination beim Hunde. [The frequency distribution of sex ratio in dogs.] *Z. Zücht.*, *B*, *36*: 227-236. [*A.B.A.*, *5*: 177.]

WITZIGMANN, J., and KÄB, E. 1937. Beiträge zur Aetiologie der Hautkrankheiten des Hundes. I. Mitteilung. Hautkrankheiten und Geschlecht. II. Mitteilung. Hautkrankheiten und Alter. III. Mitteilung. Die Beziehungen der Hautkrankheiten zu Haarbeschaffenheit und Rasse. IV. Mitteilung. Die Beziehungen der Hautkrankheiten zu Jahreszeiten und Wetter. [On the aetiology of skin diseases in the dog. I. Skin diseases and sex. II. Skin diseases and age. III. Correlation of skin diseases with coat type and breed. IV. Correlation of skin diseases with season and weather.] *Berl. tierärztl. Wschr.*: 130-132; 147-149; 333-336; 345-347. [*A.B.A.*, *5*: 426.]

WRIEDT, C. 1925. Letale Faktoren. [Lethal factors.] *Z. Tierz. ZüchtBiol.*, *3*: 223-230.

—— 1929. Aufspaltung der Schädelform des Pekingnesers (Vorläufige Mitteilung). [Segregation of the Pekingese shape of head (preliminary communication).] *Züchter*, *1*: 203-204.

WRIGHT, S. 1918. Colour inheritance in mammals. IX. The dog. *J. Hered.*, *9*: 87-90.

—— 1923. Mendelian analysis of the pure breeds of livestock. I. The measurement of inbreeding and relationship. *J. Hered.*, *14*: 339-348.

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